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Inserted in this issue of ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY *is a pink slip. It is a notification that subscriptions for the coming year are now due and payable. Those who have already paid for 1927 naturally will disregard the notice.*

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Archives of Dermatology and Syphilology

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SCLEREMA NEONATORUM *

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LONDON

In January, 1923, Dr. J. W. McNee and I read a paper before the Dermatological Section of the Royal Society of Medicine in London, describing a case of sclerema neonatorum. In this case, the patient developed a cystic swelling in one of the sclerema patches which at first was thought to be an abscess, but which when opened was found to contain broken-down fat and acicular crystals. Some interesting histologic and chemical investigations made on the fat of this infant were reported, together with some observations on the fat of an older infant who had developed a scleremátous condition as a terminal feature in a fatal attack of epidemic diarrhea. This paper was not published at the time because it was felt that, as some new facts had come to light, it would be better to wait and try to confirm them in other cases.

No further case came under observation until April, 1924, when one almost identical with our original case came under the observation of Dr. Hugh Thursfield at the Hospital for Sick Children, Great Ormond Street, and we were able to obtain the cooperation of Dr. G. A. Harrison, the biochemist at this hospital, in investigating the chemistry of the fat in this case. Dr. Harrison has further been able to obtain fat from another fatal case of sclerema in which the patient was under the care of Dr. H. C. Cameron, of Guy's Hospital, and has published his observations.[1]

REPORT OF CASES

The object of this paper is to refer chiefly to the clinical aspects of the cases; those who wish to study the chemical researches in full are referred to Dr. Harrison's papers.

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* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

1. Harrison, G. A.: Arch. Dis. Child. **1**:63 and 123, 1926; Biochem. J. **20**: 84, 1926.

The following is a description of the two cases referred to:

CASE 1.[2]—L. W. H., a boy, aged 6 weeks, was admitted to University College Hospital under Professor Choyce on Jan. 23, 1921, for what was believed to be an abscess on the left arm. He was transferred subsequently to the medical side under Professor Elliott and Dr. McNee. This was the first child of the parents. Labor was normal. The child apparently was healthy at birth. On the second day a "lump" was noticed on the upper part of the left arm. Subsequently, other "lumps" appeared in other parts of the limbs and trunk. Later the swelling on the left arm softened, and, an abscess being suspected, was opened, but only milky fluid escaped. The child was never breast fed, its food being a dried milk (Cow and Gate).

Examination.—On admission, the child was pallid and emaciated, weighing 7 pounds and 8 ounces (3.4 Kg.). Irregular, hard, well-defined thickenings of the subcutaneous tissue were present over both upper arms and over the upper part of the back and nape of the neck, and smaller areas of thickening were present over both thighs and on the chest. There was no pitting on pressure. The abdomen, forearms and hands, legs and feet were free from these thickenings. The skin was firmly adherent to these swellings and was a deep purplish color, especially in the region of the left deltoid muscle, where the swelling had undergone softening. A milky fluid could be squeezed out from an incision in this area, and, on microscopic examination, was found to contain myriads of acicular crystals, together with a certain number of cells of the lymphocytic type and fat globules. Cultures of the fluid on ordinary culture mediums remained sterile. Films examined for spirochetes by dark-ground illumination and stained by Fontana's method were all negative.

The Wassermann reactions of mother and child were negative.

Progress in Hospital.—No fresh lesions appeared during the child's stay in the hospital. The temperature was generally between 97 and 98 F., although several times falling below 97 F. On two occasions, however, a slight febrile reaction was observed, namely, 101 on January 29-30 and 100.2 F. on February 5. Several days after admission the swelling on the left deltoid region became much enlarged and definitely fluctuant; it was not dealt with surgically.

On February 3, a wedge from the swelling on the upper arm was removed for microscopic purposes.

The infant made no progress and died of asthenia on February 9, sixteen days after admission.

Necropsy (Dr. J. W. McNee).—The child was emaciated and pallid. Localized swellings in the subcutaneous tissue of the upper arms, back and back of the neck were still readily palpable in the cadaver. A fluctuant swelling over the left deltoid muscle was incised, and fully half an ordinary test tube full of milky fluid escaped. The fluid was obviously not pus. On cutting into various parts of the subcutaneous tissue of the body, the following was revealed: The subcutaneous tissue in most places was reduced to a very thin layer. In the other areas, corresponding to the palpable swellings, the subcutaneous fat was locally increased in amount and macroscopically showed various differences from the normal. It was a chalky white without a trace of yellow, and the fibrous trabeculae were sharply defined so as to give the tissue an appearance almost approaching that of the pancreas or a salivary gland. It was firm to the touch and had a peculiar lard-like consistency.

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2. Case 1, in Dr. Harrison's article (footnote 1).

The stomach was much dilated, and the pylorus distinctly hypertrophied to form a mass about the size of a lima bean. The suprarenal glands were small, and their cortex appeared to be devoid of lipoid fat. All the other viscera were normal.

Portions of skin and subcutaneous tissue were removed for histologic and chemical investigation.

CASE 2.[3]—L. H., a girl, aged 5 months, was admitted to the Hospital for Sick Children, on April 3, 1924, under Dr. Hugh Thursfield. The baby was born at full term, the labor being normal. The patient was the youngest of a family of eleven, of whom nine were healthy and one had died of marasmus. Red patches, hard and raised, were noted on the skin of the arms on the first and second days of life. On the fifth day the arms and legs were stiff. Later "lumps" appeared under the skin of the arms and legs. The weight at birth was 8½ pounds (3.8 Kg.). The baby was bottle-fed from birth. It was given a proprietary food and cow's milk in the daytime and cow's milk and whisky (10 drops to a feeding at night.

Examination.—On admission the child appeared well nourished and normal in every way, except for the subcutaneous swellings. She weighed 10¼ pounds (4.6 Kg.). Firm, hard patches, adherent to the skin and which did not pit on pressure were present in the subcutaneous tissue of both arms, both forearms, the extensor aspects of both thighs, both calves and on the back. There was a small fluid cyst over each deltoid muscle. One of these was aspirated, and about 1 cc. of an oily, pale straw-colored liquid was obtained. While drawing it into the syringe, it was noted that there were minute white lumps in suspension in the oily fluid. These lumps quickly increased in size on cooling. They did not go back into solution until the fluid was heated to nearly 50 F. On examination of stained films under the microscope, no cells and no organisms were seen. Cultures were not made. Under the polarizing microscope, the white lumps were seen to be masses of acicular, anisotropic crystals. The calcium content was 41 mg. per hundred cubic centimeters of fluid, and the total cholesterol (colorimetric determination) was 1.5 per cent, approximately.

The Wassermann reaction was negative. The feces were normal microscopically. Nothing abnormal was found in the urine.

Progress in Hospital.—On April 17, a biopsy was performed by Mr. E. J. Lloyd, who removed a portion of the skin and subcutaneous fat from the right forearm. The wound healed by first intention.

Shortly after the operation, the infant developed gastro-enteritis, from which she died on May 5. The weight remained stationary from admission until the biopsy. Thereafter it fell rapidly from 10 pounds, 7 ounces (4.7 Kg.) to 8 pounds, 6 ounces (3.8 Kg.). During this period of decline, the subcutaneous lumps softened and became absorbed to a considerable extent. The temperature from admission until the operation varied between 97 and 100 F., although it exceeded 99 on only a few occasions. After the operation until death it varied between 98 and 102 F.

Six days after the biopsy the blood was examined. The blood sugar was 67 mg. per hundred cubic centimeters half an hour after a feeding and the serum cholesterol was only 59 mg. per hundred cubic centimeters, whereas the blood of a control baby, 5 weeks old, contained 143 mg. There was no lipemia.

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3. Case 3 in Dr. Harrison's article (footnote 1).

Roentgen-Ray Examination.—A roentgen-ray examination was made, and the indurated areas were found to be studded with numerous small opacities (fig. 1).

Necropsy (Dr. W. J. Wyllie).—The body was greatly emaciated; it weighed 7 pounds (3.2 Kg.); the length was 2 feet, 2 inches (66 cm.). A small cystic swelling the size of a marble was present under the skin of each arm over the deltoid muscle. Under the skin of the forearms and of both legs were many small firm lumps. The skin over the buttocks was thick and brawny to the touch. Portions of subcutaneous fat were removed for chemical and histologic examination from the buttocks and back of the thigh and arms. Throughout the fat thus removed were scattered minute cysts similar to those over the deltoids. Each cyst was full of a white, shiny, soft, glistening material which had a strong odor resembling hydrous wool fat and camphor. The subcutaneous fat elsewhere was dry and firm. The viscera in the thorax, abdomen (apart from the intestines) and brain revealed no abnormalities.

Microscopic examination of sections of the pituitary body, thymus, thyroid, pancreas and suprarenals revealed nothing of note.

. . . . . .

Fig. 1 (case 2).—Upper limb showing position of cystic swelling and deposits of calcareous material in the subcutaneous fat.

I will here also refer to two other cases, which, though neither of them showed the cystic swellings observed in the two previous cases, have a bearing on classification of sclerema.

Dr. Cameron's Case.[4]—A boy was admitted to Guy's Hospital on April 3, 1924, when 4 days old. Labor had been normal, but the child was born with blue asphyxia. When 24 hours old, the cyanosis occurred, but there was no distress. Subsequently there were several similar but slighter attacks of cyanosis. There was diarrhea with green stools, but no vomiting. The sclerematous deposits were similar to those in case 2, but were more extensive; both cheeks were involved. The temperature fluctuated slightly on either side of 98 F., but was never definitely subnormal. On two or three occasions, it rose to 101 and 103 F. Weight on admission was 2.8 Kg., but rose to 3 Kg. when the child was discharged from the hospital on May 7, 1924.

The infant was readmitted on July 2, 1924, with infective enteritis, from which he died the next day.

4. Case 4 in Dr. Harrison's article (footnote 1).

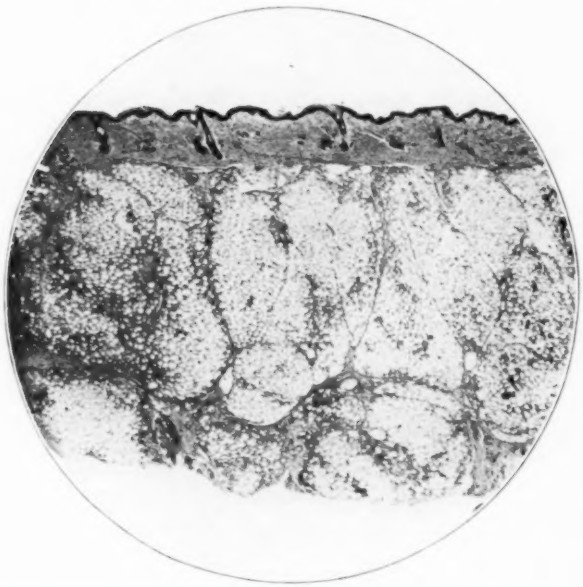

Fig. 2.—Low power section of skin and subcutaneous fat showing cellular infiltration into fat lobules, most marked on the left side of the section. The dark staining areas to the extreme right of the section are calcareous deposits

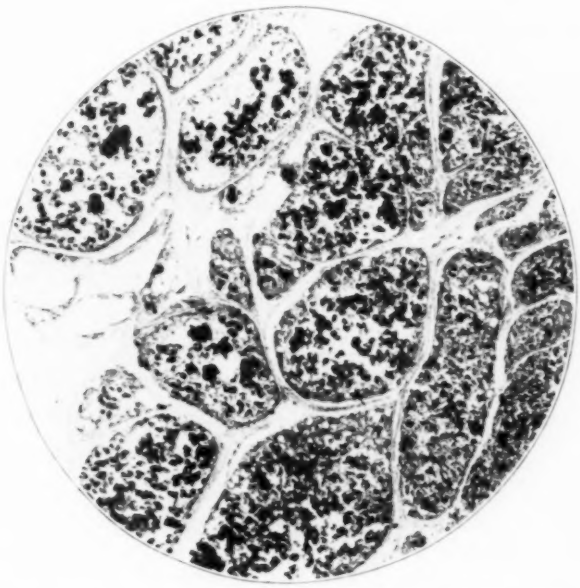

Fig. 3.—Low power section of subcutaneous fat stained with sudan III, showing disintegration of liquid fat and involvement of fat lobules by cellular infiltration.

A roentgenogram failed to reveal the shadows shown in case 2, but as the calcium deposits were found histologically, it is possible that too hard rays were employed.

Necropsy.—This revealed nothing abnormal with the exception of the condition of the intestines and subcutaneous fat.

Dr. Thursfield's Second Case.[5]—A girl, aged 6 weeks, was admitted to the Hospital for Sick Children on Nov. 11, 1924. She was born at full term. Labor was normal. Weight at birth 7 pounds (3.2 Kg.). Hardness of buttocks was noticed during the first week of life, then shoulders, arms, back and thighs became hard. The child was admitted on account of "fits" and "blue turns" which also started during the first week of life. The sclerematous deposits had the same characters as those described in the previous cases; the cheeks were not involved. The Wassermann reaction was negative. Nothing else abnormal was found.

The infant slowly gained weight and improved generally, the sclerematous deposits slowly absorbing until recovery became complete in the sixth month. At the age of 10 months, the infant still had fits, though these were less severe and frequent. It was doubtful whether the child had ever been able to see, although nothing abnormal was found on examining the fundi and optic disks. A roentgenogram of the skull showed no abnormality.

Roentgen-Ray Examination.—Examination of the subcutaneous fat showed the same kind of shadows as were found in case 2. These shadows persisted until long after the subcutaneous thickenings had disappeared. On Jan. 2, 1925, they were little altered, and in February, 1925, they were still visible, but appeared less marked. In July, 1925, no opacities were visible in the roentgenograms.

No biopsy was made.

HISTOLOGY

Portions of skin and subcutaneous tissue obtained both by biopsy and at necropsy in cases 1 and 2 were hardened in a 10 per cent solution of formaldehyde and cut in gum and paraffin. The preparations were made under the direction of Dr. McNee, who has described the changes found in the paper by Dr. Harrison, to which reference has previously been made.

The epidermis and dermis were normal. The most striking change noted was a marked thickening of the trabeculae between the fat lobules, which was so marked as to be visible macroscopically. From these trabeculae masses of cells could be seen invading the surrounding fat lobules (fig. 2). These cells were chiefly of the fibroblastic type, but numerous giant cells of irregular shape were seen, as well as a few groups of lymphocytes and a few leukocytes. These cell masses were seen to be penetrating into the septums between the fat cells, a large number of which had been entirely absorbed (fig. 3).

A curious change was visible in some of the empty fat cells seen in paraffin sections. These empty cell spaces appeared to be bridged across in many places by strands of protoplasm from the wall of the

5. Case 5 in Dr. Harrison's article (footnote 1).

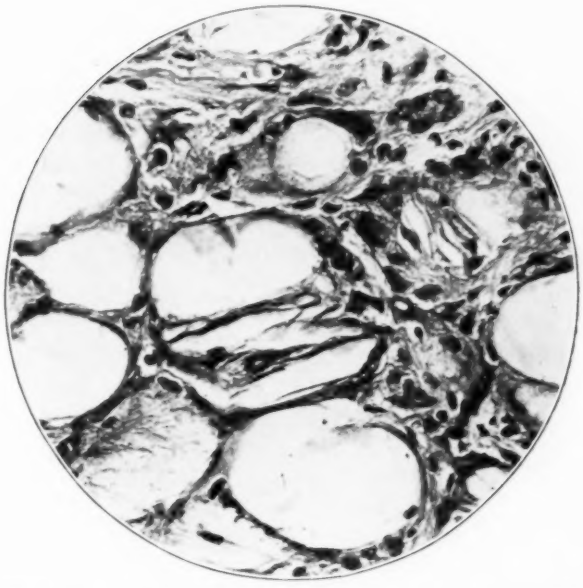

Fig. 4.—High power section showing type of cellular infiltrate, fibroblasts and foreign-body giant cells. Protoplasmic masses are also seen invading the fat cells, and in these, clefts left by dissolved out crystals are visible. These clefts are also seen in the cell masses.

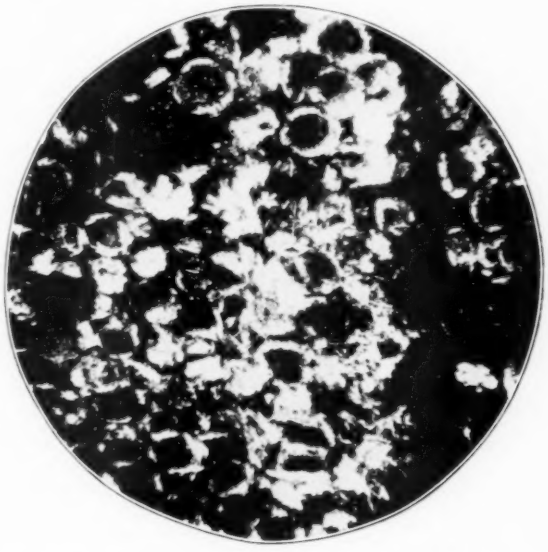

Fig. 5.—Low power section seen through crossed Nicol's prisms. The doubly refracting crystals are seen to shine up brightly throughout the section.

cell, leaving narrow clefts between the strands. In the cellular exudate around the cells, similar clefts could be seen (fig. 4).

In sections cut in gum and stained with sudan III, these clefts contained acicular crystals which did not stain with "sudan" and were anisotropic, that is to say, shone out brightly when examined with crossed Nicol's prisms (fig. 5). These crystals, present in enormous numbers in the sections, were not only present in the clefts in the cellular exudate, but were also present in relation to the fat cells not surrounded by these invading cells. They were found in cells which stained well

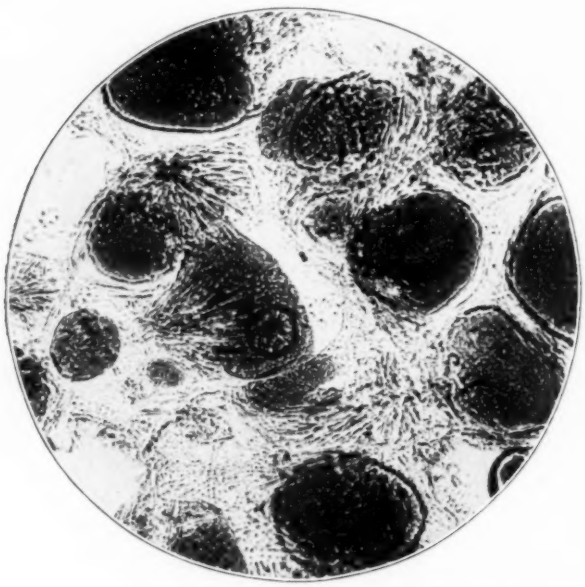

Fig. 6.—High power section stained with sudan III, showing deposits of crystals in the fat cells.

with sudan, but were also found in masses or rosets in fat-cell spaces which took little or no stain with this dye (fig. 6).

In some parts of the sections, mostly in those parts in which there was no cellular infiltration, there was a well-marked deposit of lime salts, recognized by their density, shape and deep purple staining to hemalum. This explains the shadows seen in the roentgenograms in case 2.

A further change noted was some thickening in the walls of the smaller arteries.

The appearances described above were found to be present in a similar degree in Dr. Cameron's case at Guy's Hospital already referred to.

CHEMISTRY OF THE SUBCUTANEOUS FAT

Chemical examination of the fat in the first case was made by Dr. McNee, in the second and in Dr. Cameron's case, by Dr. Harrison. The latter has, however, had greater opportunities of investigating the subject, and the following results are mainly a summary of his observations.

Nature of the Crystals.—Dr. Harrison first attempted to determine the nature of the crystals found in such large quantities in the sections. These crystals had previously been noted by Knöpfelmacher [6] and others, but it had always been assumed that they were fatty acid crystals. It is also known that in histologic preparations of normal fat, crystals may sometimes be found in the fat cells. Dr. Harrison finds that the solubilities of the sclerema crystals exclude the possibility of their being soaps or urates. The low acid values of the scleremato us fat (to be referred to later) makes it unlikely that the abundant sclerema crystals are free fatty acids, although it is noted that the acid values of scleremato us fat are slightly higher than those of nonscleremato us fat. The fact that they are only slightly soluble in warm alcoholic solution of alkali, however, rules out the possibility of their being free fatty acids.

He does not consider that they can be composed of cholesterol esters, for the following reasons: (*a*) Microchemical tests for cholesterol were negative. (*b*) On carefully warming the crystals under the polarizing microscope and then cooling, no evidence of the formation of fluid crystals was ever noted. (*c*) The melting point of the sclerema crystals was lower than the melting point of the true cholesterol esters (i. e., as seen in frozen sections of cutaneous xanthoma). (*d*) Had the sclerema crystals been cholesterol compounds, one would have expected, because of the large quantity of crystals seen in the sections, that the cholesterol content of the ether extracts would have been higher than that actually found (i. e., case 1, at necropsy, 0.45 per cent; case 2, at biopsy, 1.5 per cent, at necropsy, 6.0 per cent; Dr. Cameron's case, at necropsy, 2.1 per cent).

The solubility of the sclerema crystals in acetone makes it unlikely that they are phosphatides or cerebrosides. The presence of the latter compounds is also negatived by the absence of liquid crystal formation (Maltese crosses). Also the residue obtained after extraction of the crystals with ether, yielded no ash on ignition. It is concluded therefore that in all probability the sclerema crystals are neutral fats—either palmitin or stearin, or both, or the glycerides of higher fatty acids.

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6. Knöpfelmacher, W.: Jahrb. f. Kinderheilk. **45**:177, 1897.

Experiments made with pure palmitin crystals showed exactly the same solubilities as crystals found in nonsclerematous fat, the sclerema crystals differing only by being insoluble in cold petroleum ether, though soluble in this substance when warm.

Dr. Harrison also points out that there is a general assumption among histologists that anisotropic crystals are cholesterol esters or "cholesterol fatty acid mixtures." He shows, however, that, while liquid fat is isotropic, the crystals of neutral fats and fatty acids are invariably anisotropic.

Melting Point of Crystals.—Experiments were then made as to the melting points of the crystals. It was found that the sclerematous crystals melted at from 40 to 50 C. The crystals melted suddenly when the critical temperature was reached and reappeared with equal suddenness when it again fell below this point. Similar observations were made on nonsclerematous fat removed from infants at necropsies or operations. It was found here that the crystals melted at about 30 C. and reappeared more slowly at room temperature. It was further noticed that in the latter case the crystals were confined to the fat cells and were not found in the cellular tissue as described in sclerematous cases. When, however, the ether extract of the crystals of nonsclerematous fat was examined, the melting point was found to be the same as for the ether extract of sclerematous crystals, namely, 40 to 50 C.

Experiments were then made to determine whether the melting point of the crystals varied with the percentage of palmitin present, and it was found that if 3 per cent of palmitin was added to an ether extract of nonsclerematous subcutaneous fat, the melting point was raised 8.9 C.; when 7.1 per cent was added, the melting point was raised 17 C. On the other hand, the addition of 2 per cent of cholesterol only raised the melting point 0.1 C.

It follows from this that a slight diminution of olein present in the fat would increase the percentage of palmitin and thus materially raise the melting point of the fat.

Observations on the Olein Content of the Fat.—Dr. Harrison examined the fat of a series of eight nonsclerematous infants and children and one adult, and confirmed the observations of Knöpfelmacher,[6] Thiemich,[7] Siegert,[8] Jaeckle,[9] Bardisian,[10] Finkelstein and Sommerfeld,[11] Lasch [12] and others that the olein content of an infant's fat is less than

7. Thiemich, M.: Ztschr. f. Physiol. Chem. **26**:189, 1898.
8. Siegert, F.: Beitr. z. chem. Physiol. u. Path. **1**:183, 1902.
9. Jaeckle, H.: Ztschr. f. Physiol. Chem. **36**:53, 1902.
10. Bardisian, A.: Pediatria **29**:159, 1921.
11. Finkelstein, H., and Sommerfeld, P.: Monatschr. f. Kinderh. **25**:105, 1923.
12. Lasch, W.: Jahrb. f. Kinderh. **107**:223, 1925.

that of an adult's. He also finds that the iodine value of the ether extracts of scleromatous fat is slightly lower than that of the fat of nonscleromatous infants of corresponding ages.

The following table shows the iodine values and melting points of samples of fat from the two cases reported above and from Dr. Cameron's case, compared with the fat of nonscleromatous infants of approximately the same age.

Iodine Values and Melting Points of Samples of Fat

	Age, Weeks	Iodine Value		Melting Point	
		Sclerema	Normal	Sclerema	Normal
Case 1 (necropsy)	6	42.1	.....	50.5	.....
Case 2 (biopsy)	21	52.3	56.9-60.6	38.5	30.5-27.9
Case 2 (necropsy)	24	54.0	56.9-60.6	42.9	30.5-27.9
Dr. Cameron's case (necropsy)	12	45.5	53.8	47.3	37.3

It will be seen that there is a definite though small diminution in the iodine values in scleromatous as compared with nonscleromatous fat, and that this fall corresponds with a rise in the melting points. If it is assumed that olein is the only unsaturated fat present in the tissue, an assumption that would appear to be justified in the control cases, though not so certainly in the sclerema cases, then a rise or fall in the iodine value should represent a rise or fall in the amount of olein present.

Calcium Salts.—Finally Dr. Harrison endeavored to find out in what form the calcium, shown in the histologic preparations and also in the roentgenograms, was present. He was able to exclude the presence of soaps in the ether extracts of the fat, but found an excess of phosphorus in the tissues in even greater quantities than would be necessary to combine with all the calcium present to form basic calcium phosphate ($Ca_3(PO_4)_2$).

THE NATURE OF THE CHANGES IN THE FAT

In order to come to any conclusion as to the sequence of changes occurring in the fat in these cases, it is necessary to take into consideration both the histologic and the chemical changes observed. The appearance of the sections, especially those stained for fat, suggest that the earliest changes take place in the fat cells themselves. Something occurs which causes crystals to appear in a number of the fat globules. Following this a tissue cell reaction takes place, commencing in the trabeculae between the fat lobules, with the object of absorbing these crystals which presumably act like foreign bodies and eventually replacing the destroyed fat cells by new connective tissue.

That this is the sequence of events is probable from the fact that the crystal formation in the fat cells takes place more or less generally throughout the fat lobules, and is not confined to the periphery of the

lobules where the cellular reaction is most marked. Further, the type of cellular exudate, consisting of fibroblasts and the so-called "foreign-body" giant cells, is indicative of what one may call a "mopping-up" process.

If one looks at the least affected fat cells, it is, I think, possible to observe a small tuft of crystals developing in one part of the fat globule, close to the cell wall. Later the crystals fill the cell, and the staining of the cell contents is fainter, doubtless due to the diminution of the liquid fat in the cells. Still later the staining disappears, and the outline of the cell can be made out surrounding a colorless roset of crystals; finally, these crystals can be found free in the tissues.

We have now to consider what causes these crystals to form in the fat cell. It is clear, as is mentioned above, that the liquid fat is gradually absorbed from the cell. As the crystals are probably palmitin, and possibly stearin as well, this means that the olein is gradually absorbed, leaving the higher fats behind in crystalline form. Probably this is what happens from the first: a slight reduction in the amount of olein in the fat will bring about a relative increase in the percentage of the palmitin and stearin present, and this, as Dr. Harrison's experiments show, will cause a great rise in the melting point, so that a point will soon be reached at which the higher fats will crystallize out at body temperature. It is unnecessary for the body temperature to be lowered to bring about this crystalization, as it could take place even with fever, if the olein were sufficiently diminished; and we assume in these cases that it is eventually absorbed completely in many of the fat cells.

These views would appear to be in keeping with those of Langer,[13] expressed in 1881, that some diminution of the olein in the fat occurs (he suggested a preferential oxidation of the olein, but this is purely theoretical). He also considered a lowering of body temperature was essential, but, as has been pointed out, this is not so.

Bayer [14] also supported the view that diminished oleic acid was the cause of sclerema.

In opposition to this is the view expressed by Siegert,[8] that the diminished olein hypothesis could not be held in view of the observations of Thiemich [7] that there was no significant difference between the iodine values of the fat of two sclerematous infants and that of normal infants of the same age. Miura [15] also concluded that the fat of sclerematous infants did not differ materially from the fat of normal infants. Sarvonat [16] came to much the same conclusion. Smith [17] also

13. Langer, L.: Wien. med. Presse **22**:1375 and 1412, 1881.
14. Bayer, R.: Verhandl. d. deutsch. path. Gesellsch. **12**:305, 1908.
15. Miura, quoted by Smith: Japan Ztschr. f. Dermat. u. Urol. **5**:412, 1905.
16. Sarvonat, quoted by Smith: Arch. de méd. d. enf. **9**:22, 1906.
17. Smith, C. S.: J. Cutan. Dis. **36**:436, 1918.

found the same, but his experiments on normal fat were taken from what appeared macroscopically to be normal in a sclerematous infant. This, however, may give fallacious results, as it has been shown in the cases described above that fat, apparently normal, may show definite sclerematous changes microscopically.

It has further been shown by Dr. Harrison that it is not necessary to get a marked fall in iodine value to cause a considerable raising of the melting point of the fat. It may be further noted here that, as far as can be ascertained, none of these observers controlled their chemical observations by microchemical tests, and it would seem from the foregoing that there must be gradual absorption of the olein from the fat cells.

The question as to what causes the primary changes in the fat is, I believe, impossible to answer at the present time. It has been suggested that it is due to the lowering of body temperature as the disease occurs in weak, debilitated infants. If the changes were simply those which are produced by cooling fat below its melting point, it is conceivable that there might be something to be said for the argument, in view of the fact that it is generally accepted that the fat of new-born infants melts at about normal body temperature; but the histologic facts are otherwise.

Further, although in a few cases of sclerema there may be markedly subnormal temperatures, it is certainly not the rule. In the four cases mentioned above, the temperatures were practically always above 97 F. I thought it would be interesting to determine what was the "normal" temperature of new-born infants, and for that purpose I have examined the charts of fifty healthy babies delivered consecutively at University College Hospital. I found that in twenty of these (40 per cent) it was constantly between 97 and 98 F. during the first ten days of life. It may, therefore, be concluded that a temperature down to 97 F. can scarcely be considered subnormal in a new-born infant.

The distribution of the eruption is a further argument against the temperature theory, for the fat is not uniformly affected. The fact that the lesions appear as small isolated nodules is also not what one would expect in these circumstances.

Trauma during labor or during artificial respiration has also been suggested as a cause of the fat changes. If this was the only cause, it should occur with much greater frequency than it does, and one would not expect a symmetrical distribution of the lesions. It is also clear that many cases have not been exposed to severe handling.

Ballantyne,[18] in his admirable survey of the literature of sclerema up to 1895, gives an account of a number of theories that have been

18. Ballantyne, J. W.: The Diseases of the Foetus, Edinburgh, vol. 27, 1895.

held, in addition to that mentioned above. Apart from the inflammatory theories, these do not require serious consideration in the light of modern pathologic knowledge.

The two most probable theories are either some congenital disturbance of fat metabolism or some toxic influence causing damage to the fat cell. It is possible to conceive of some congenital tendency to fat splitting, but it is difficult to find any evidence in favor of this view. It would seem more probable that some toxic influence is at work to damage the fat cell. In this connection it may be noted that Smith [17] considered that the change taking place (as far as he determined it) might be similar to that seen in fat necrosis. He bases his view largely on the high fatty acid content of the fat as determined by the acid value. He obtained an acid value of 79 for the "normal" fat of the sclerematous infant and 163 for the sclerematous fat. Unfortunately, in no other cases of scelema has the acid value been determined except in case 2 reported above and in Dr. Cameron's case. In these two Dr. Harrison found the following acid values: Case 2: biopsy 5.4, necropsy 9; Dr. Cameron's case: necropsy, 5.9. In a series of control necropsies of infants under 1 year of age, the acid values varied from 0.6 to 2.7. The difference in Dr. Smith's figures is so great as to suggest that, in his case, postmortem hydrolysis had taken place.

It must be admitted, however, that the descriptions of the microscopic examination of fat necrosis resemble closely those seen in sclerema. There is the disappearance of the liquid elements of the fat, with deposition of rosets of acicular crystals and also the deposit of calcium salts. Wells [19] states that these crystals are fatty acid crystals as they stain with Benda's stain (a copper acetate mixture which stains fatty acid crystals, but not those of neutral fat). Unfortunately, this test was not employed in our cases, but in a recent paper by Carol and Van Der Zande,[20] sections of a sclerema case were stained with a similar stain (Fischler's copper-corrosive-hematoxylin) with a negative result, thus confirming the view that the crystals in sclerema are not fatty acids. These observers, however, concluded that the crystals were cholesterol esters, chiefly on microscopic tests. They did not carry out the exhaustive tests for cholesterol as did Dr. Harrison. It is probable therefore that we are dealing with a different process than that encountered in pancreatic fat necrosis.

Of possible bacterial toxins, syphilis has been considered a cause of the disease. In our cases, however, this may be ruled out. No organ-

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19. Wells, H. G.: Chemical Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1925.

20. Carol, W. L. L., and Van Der Zande, F.: Nederl. Tijdschr. v. Geneesk. **69**:1317, 1925.

isms of any kind were discovered, but it is obvious that their presence cannot be completely excluded.

There seems to be nothing further to add at the present time to the possible causes of the fat changes, this must be left to future investigations.

OTHER REPORTED CASES

The only way in which the two cases described differ from Dr. Cameron's case and Dr. Thursfield's second case is in the presence of cystic swellings from which were obtained fat and acicular crystals. It is by reason of the rarity of this complication that special attention has been called to them. This condition is not recorded by Ballantyne,[18] who has analyzed all the published cases up to 1895, but it has been present in at least three cases, published since then.

In 1907, Fabyan [21] published a case in the *Johns Hopkins Hospital Bulletin* of an infant who developed numerous "abscesses" on both cheeks, both forearms, back of the head, one buttock and the right lower leg. All these, except the one on the right cheek, "absorbed without suppuration." Pus was removed from the right cheek. The child died of suffocation on the fourteenth day.

Histologic examination showed granulomatous changes similar to those observed in our cases, and the clefts in the cells were noted which the writer attributes to spaces left by fatty acid crystals, although he does not appear actually to have observed the crystals. The case is described as one of "disseminated subcutaneous fat necrosis occurring in an infant without other lesions."

At a meeting of this Association in May, 1917, the late Dr. F. G. Harris,[22] discussing a paper by Dr. Lieberthal, entitled "Sclerema Neonatorum and Scleroderma," described a similar case as follows:

> The first case (of sclerema neonatorum) he saw began in the same way. It looked like an angioma, spread over the arms and down the posterior surface of the thighs. The patient ran a temperature of 101°-105° F. for six weeks, although previously to that time he was perfectly well. The condition began about a week after birth. The child was nursed and grew, and a peculiar thing was that over the area involved the lesions became soft. There was a semi-fluctuating mass of sebaceous-looking material containing globules of oil, which under the microscope was found to be composed of needle-shaped crystals.

The third case was published in 1922 by Keilmann,[23] under the title of "Circumscribed Symmetrical Fat Sclerosis in an Infant (the So-Called Scleroderma)." The infant was first seen when 11 days old, with swelling on both cheeks, subsequently developing them on the chin,

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21. Fabyan, M.: Bull. Johns Hopkins Hosp. **18**:349, 1907.
22. Harris, F. G.: Discussion of Lieberthal's article, J. Cutan. Dis. **36**:29, 1918.
23. Keilmann, K.: Ztschr. f. Kinderh. **33**:298, 1922.

both arms and thighs, middle of back, over the scapulae and buttocks and small areas on the forearms and abdomen. An "abscess" developed on the right cheek, which was thought to be due to infection from the mouth, but it subsided apparently without being opened. The child died on the 119th day of osteomyelitis, and a detailed histologic examination of the patient was made by Busse, which corresponds closely with that already described. The crystals were noted, but apparently no chemical observations were made. I think there can be no doubt that the case is similar to those described, but it is possible that the swelling was a true abscess.

NOMENCLATURE

There still appears to be considerable confusion as to the nomenclature of these cases. The amount of literature on sclerema published prior to about 1900 is enormous and most conflicting, and this confusion is still evident in some of the writings. Ballantyne [18] and others have, more or less successfully, tried to bring order out of chaos, but I think it is wise to disregard, for the present, all the older literature and see how things stand in the light of modern knowledge.

The condition known as edema neonatorum appears to be a true edema occurring in the new-born, is easily recognized as such both clinically and histologically, and need not be considered in connection with our present subject. I imagine that such cases are rarely seen by dermatologists.

Scleroderma is a well-defined condition. In its diffuse form, the only form likely to be confused with sclerema, it must be extremely rare in new-born infants, although some genuine cases appear to have been published. Carol and Van Der Zande [20] quote cases by Löwenberg and Kneschke as true examples. The condition cannot be confused if a biopsy is made, as the changes in scleroderma are mainly seen in the collagen bundles and there are no primary fat changes, although the fat cells may be destroyed by encroachment of fibrous tissue. It is true that a few anisotropic crystals may be found in the fat cells, as they may be in most sections, but these numbers are so few as to be readily distinguished from the enormous masses found in sclerema, and they are confined to the fat cells. Also the liquid fat is unaltered. Clinically, too, diffuse scleroderma does not occur in sharply defined plaques and nodules, but in diffuse, ill-defined areas.

It is necessary to make this mention of scleroderma because some cases of sclerema are described under the term "scleroderma neonatorum," for example, the cases of Keilmann,[23] Bernheim-Karrer [24] and others. It is clear that these are cases of what we call sclerema, and it is unfortunate that this term should persist.

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24. Bernheim-Karrer: Schweiz. med. Wchnschr. **3**:12, 1922.

When we turn to the cases in which the main feature is a consolidation of the subcutaneous fat, there appears to be no doubt that we have two types of cases to deal with.

The first type of case is the one to which the cases already referred to belong. The process of solidification of the fat is more or less gradual and almost invariably commences in the first few days of life. It is questionable whether it occurs after the first month, and, although a good number of cases have been described in which the condition was present at birth, it would be as well to reserve judgment until further investigations have been made on such cases. The disease attacks infants who generally appear healthy, whose temperature is not markedly subnormal, who, as often as not, are born of healthy parents and have received no injury at birth nor have been born with severe asphyxia. The regions chiefly affected are the cheeks, shoulders, scapular regions, arms, buttocks, thighs and calves. A considerable number of patients recover, and those who die frequently do so from some intercurrent malady.

It is to this type of case that we, in England, usually apply the name sclerema neonatorum, and I think it is the most appropriate name for the condition, as it is short and does not commit one to any pathologic theory as to causation. It is to this class of case that the term "pseudosclerema" has been applied by some pediatricians, and I think it is unfortunate that confusion should have been introduced by the invention of this title. Also to the same class belong the cases described recently as "subcutaneous fat necrosis," "subcutaneous fat sclerosis," "adiponecrosis subcutanea neonatorum," etc. These terms are unwieldy and presuppose a definite pathologic process, which is still open to argument.

The second type of case appears to occur in weakly, premature or marasmic infants or in those who are more or less moribund from some severe wasting disease, especially from epidemic gastro-enteritis. The solidification of the fat occurs with much greater rapidity than in the former type and is much more diffuse, the whole of the body surface being affected, with the exception of the penis, scrotum, palms and soles. It may occur at any time during the first six months of life and is more common during the summer months. The following is an example of this type of case, the notes of which were obtained for Dr. McNee and myself by Dr. Donald Paterson, who also obtained some of the fat at necropsy for microscopic and chemical investigation.

REPORT OF CASE [25]

A girl, aged 12 weeks, was admitted to Great Ormond Street Hospital under Dr. Robert Hutchison on Sept. 22, 1921, with a three days' history of diarrhea, but no vomiting. The baby was born at full term, the labor being normal. On

25. Case 2 in Dr. Harrison's article (footnote 1).

examination there was marked dehydration and a slight enlargement of the liver, but otherwise nothing abnormal. The temperature fluctuated between 98 and 103 F. Death occurred on September 29 from ileocolitis. Two days before death the subcutaneous tissues became hard and boardlike (body temperature, 103 F.). On the next day the sclerematous deposits were more marked. There was no pitting on pressure.

Necropsy (Dr. Donald Paterson).—The body was that of a fairly well nourished child, but the subcutaneous tissues were hard and firm as a result of the condition of the sclerema. The weight was 8 pounds, 10 ounces (4 Kg.), and the length 23 inches (58 cm.). Both small and large intestines had a flannel appearance throughout. Petechial hemorrhages were present, and the colon was definitely thickened. No ulcers were seen. The mesenteric glands were much enlarged. The liver had a fatty appearance. The kidneys exhibited cloudy swellings and fatty degeneration, and uric acid infarcts were noted. Dr. Nabarro isolated *B. dysenteriae* (Flexner Y) from a scraping from the colon.

The case differs from the others described both histologically and chemically. No fat crystals were observed, and although there was a slight increase in cells in the regions of the trabeculae between the fat lobules, the sections differed very little from those of normal fat. No calcium deposits were found. Dr. McNee found, on chemical examination, that the melting point of the fat was 38.5 C., a figure only slightly above normal, and that the iodine value was 52. This suggests that the fat changes are different from those found in sclerema neonatorum cases, but, before drawing any definite conclusions on the nature of these changes, an investigation of further cases is desirable.

It would appear that both on clinical and pathologic grounds, these cases should be differentiated definitely from sclerema neonatorum. Ballantyne [18] held this view as a result of clinical observations only. It is possible that it was to this type of case that the name sclerema was first applied, but it is obviously wrong to apply the term sclerema neonatorum, as it may commence after the first few weeks of life. To call it "true sclerema" in contradiction to "pseudosclerema" also seems bad to me. The older term "sclerema adiposum" might equally well be applied to both types. A new term is required to distinguish this type of case, and I would, with diffidence, suggest that "acute sclerema" would be appropriate.

Lastly, a word must be said about the term "scleredema." This is used by some to indicate "edema neonatorum," by others to indicate an edematous form of "sclerema." Whether either type of sclerema ever exhibits an edematous form, I am not in a position to say, but if it is so, it would be preferable to use the term "edematous sclerema" and to drop "scleredema," which must inevitably lead to confusion.

In conclusion, I desire to express my thanks to Dr. McNee and to Dr. Harrison for allowing me to record the results of their investigations, and to Dr. Thursfield and Dr. Cameron for permission to record their cases; also to Dr. Donald Paterson for his notes on the case of "acute sclerema."

DISCUSSION

DR. RICHARD L. SUTTON, Kansas City, Mo.: I have never seen a case of sclerema neonatorum, but what I particularly admire is Dr. Gray's clean-cut differentiation of the condition.

DR. DAVID LIEBERTHAL, Chicago: I should like to ask Dr. Gray whether any quantitative estimations of the fats were made in his cases. According to Knoepfelmacher, the percentage of olein in the beginning of the first year of the normal infant is very low and gradually increases until it reaches normal values, as in adults, in the twelfth month.

The differentiation between sclerema and scleroderma may be difficult on rare occasions; and here the histologic examination would have to decide. Up to 1917 there were published about thirty cases of scleroderma in infants, including my own. In these publications, as I remember, little is mentioned of histologic examinations. I may add that Kneschke reported a case in the *Archiv* of 1923, in which an account of the histology is given.

DR. FRED D. WEIDMAN, Philadelphia: I wish to join my two colleagues in expressing admiration and appreciation of this excellent paper, particularly as it fills, in a most satisfactory way, one of the many gaps in the chemical examination of these cases. Last year, Dr. Hart-Drant presented a case of sclerema before our Philadelphia Society in which the patient recovered entirely and the symptoms disappeared. At birth, the child needed resuscitation and was soundly slapped, following which the lesions (which were markedly erythematous and infiltrative) developed. The illuminating feature was the histologic picture, which was a replica of what Dr. Gray has shown—degenerate fat cells and also the foreign body giant cells, plus an early fibrosis. In this case I think it was the fibrosis which was fundamentally responsible for the inflammatory and tumid condition rather than a disturbance in chemical composition of the fat. I am very much in sympathy with the hint that sclerema neonatorum is not an etiologic entity, and that there may be various morbid factors operative which direct our attention to these classes clinically.

DR. WALTER J. HIGHMAN, New York: I wish to ask a question for my own information. Normally in the skin of infants there is a papillary layer of the corium, and then a very thin horizontal layer of fat; underneath this there is a very broad layer of connective tissue and then the hypoderm. Instead of the two layers as found in the adult, there are four layers, two of fat and two of connective tissue alternating. Is the deposit Dr. Gray has mentioned found in the interpolated layer or in the usual layer? I think that this interpolated layer which vanishes within a few months may be particularly vulnerable, and that perhaps if some special examination is made of this layer something may be found which may throw light on the pathogenesis of the condition as a whole.

DR. CHARLES J. WHITE, Boston: I shall have to revise my ideas, for I had always supposed that the cases of sclerema neonatorum were always accompanied by subnormal temperature. We had a remarkable instance in the Massachusetts General Hospital several years ago in which the baby looked like a small dressed pig that had just been taken out of the icebox. Shortly after that Dr. Cushing read a paper on the hibernating animal, showing that hibernation with its lowered temperature was due to pituitary alteration. On that idea I worked up a theory that these children had some pituitary disurbance, the same as hibernating animals, and as a result of this dysfunction they had sclerema neonatorum.

I should like to ask whether the skull was opened in Dr. Gray's cases, and whether any change in the pituitary gland was found. No examination was possible in our case.

Dr. John H. Stokes, Philadelphia: I should like to ask whether Dr. Gray has noted any relation between congenital syphilis and sclerema neonatorum. I ask this because the late Dr. Frederick G. Harris had the opinion that patients with sclerema neonatorum often had congenital syphilis. In two cases I have seen, syphilis was present, and in another instance the syphilitic mother was given permission to become pregnant and bore a child with sclerema neonatorum. This child recovered promptly under inunctions.

Dr. A. M. H. Gray, London: I feel that an apology is necessary because the chemical work, which is so important, has been done mainly by Dr. Harrison, but we felt that this was a subject that should be brought before dermatologists, and as Dr. McNee and I started the ball originally it was decided that I should bring this matter before you.

The quantitative estimation of olein has been worked out, and will be brought out in full in a paper by Dr. Harrison.

Regarding the cases of scleroderma, it seems to be proved that infantile scleroderma does exist, as in Kneschke's case, but it is also certain that many cases of sclerema have been described as scleroderma.

Perhaps we see more of these cases in England than you see here I think we see more mild cases than severe ones, and I believe, and this is borne out by reading the paper of Bernheim-Karrer, that some mild cases occur which are overlooked. Also, the theory that this disease occurs only in the malnourished children is wrong. Several cases have occurred in private practice in well nourished children.

I cannot answer Dr. Highman's question offhand. Perhaps he would like to look at some of the sections and make up his mind about it.

Replying to Dr. White, I believe the head was opened but nothing was reported. As to the pituitary gland, I cannot say how carefully it was examined, but the other glands were investigated.

Replying to Dr. Stokes' question as to congenital syphilis, I think we can exclude it in these cases of ours. The Wassermann reaction in both mothers and children was negative, and we could find no evidence of the disease.

I can add nothing to the question of etiology. The actual cause of the fat changes, I think, must remain open for further investigation.

DISSEMINATED PALMAR AND PLANTAR KERATODERMA

REPORT OF A CASE *

DAVID LIEBERTHAL, M.D.
CHICAGO

To the twenty-seven cases of multiple circumscribed keratoses of the palms and the soles without a known etiology that have so far been reported in the literature, another should be added, not only on account of the rarity of the condition, but also because it has been observed over a long period. These cases form a separate group because of their identical clinical and anatomic manifestations, their course and the futility of all treatment. No uniform and appropriate name has so far been proposed for them, as various authors have chosen different ones.

REPORT OF CASE

A man, aged 58, a broker, was first seen by me in October, 1903, for a condition of the hands and feet which had begun fifteen years previously. He had not suffered from any other skin disease or serious illness. He did not feel itching or pain in these parts but was conscious of the hard nodules when bathing. No other member of his family was suffering from this disease except a brother, two years his senior, whose feet had been affected since youth. The examination of the patient disclosed no pathologic condition except that of the feet and hands. On the plantar surfaces of some toes, especially the large ones, on the balls and heels, and somewhat above these, there were numerous, dark yellow, round, horny, flat elevations, varying in size from that of a pinhead to that of a pea, with pointlike and larger central depressions. On the palmar surface of the hands and some fingers were just a few lesions. There was also in the palm of the right hand a group of whitish pinhead-sized papules. In no place was any diffuse keratotic area found. All the lesions were circumscribed, roundish, depressed in the center and implanted in perfectly normal skin. There was no erythematous area around them.

The condition of the patient yielded only moderately to treatment. Salicylic acid preparations, cauterization and other methods helped to soften and flatten but did not remove any of the horny growths. The roentgen ray produced no result.

I saw the patient thereafter from time to time, and the condition of his feet was growing steadily worse, until it caused great discomfort when he was walking. A chiropodist pared off the horny lumps whenever they grew worse. The last time I saw him was in the spring of 1925, when I examined his hands and feet again. The lesions of the hands had remained practically the same

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* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

as at his first visit in 1903, except that the group of pinhead-sized papules in the right palm had disappeared. The plantae showed a larger number of lesions, and many of the old ones had grown quite large—up to the size of a bean, especially on the heels. Those on the plantar surface of the toes had grown larger. Nowhere had any coalescence of lesions taken place. They showed the same characteristics as the lesions first seen, except that the larger

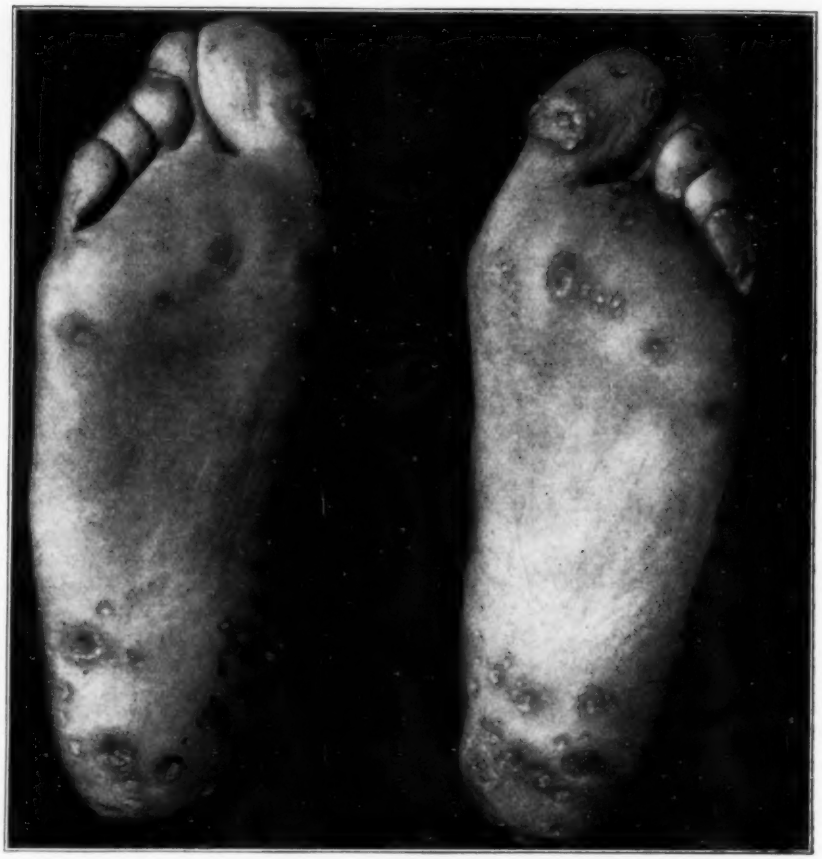

Fig. 1.—Lesions on the soles.

ones were slightly more elevated and their central depressions had become larger and crateriform.

During the whole course of the disease the patient had not suffered from hyperhydrosis, nor were the nails of the fingers, or of the toes, in any way affected. A number of nodes were at this time trimmed down deeply, and radium applied, but no result was noted. The patient was then instructed to take prolonged daily foot baths in soap suds and to apply salicylic soap plaster.

This treatment softened and flattened the growths so that he could walk comfortably. On the photograph of the right heel the effect of this treatment can be seen.

At the occasion of a visit in 1918 a small nodule was excised from the palm for pathologic examination, which gave results identical with those

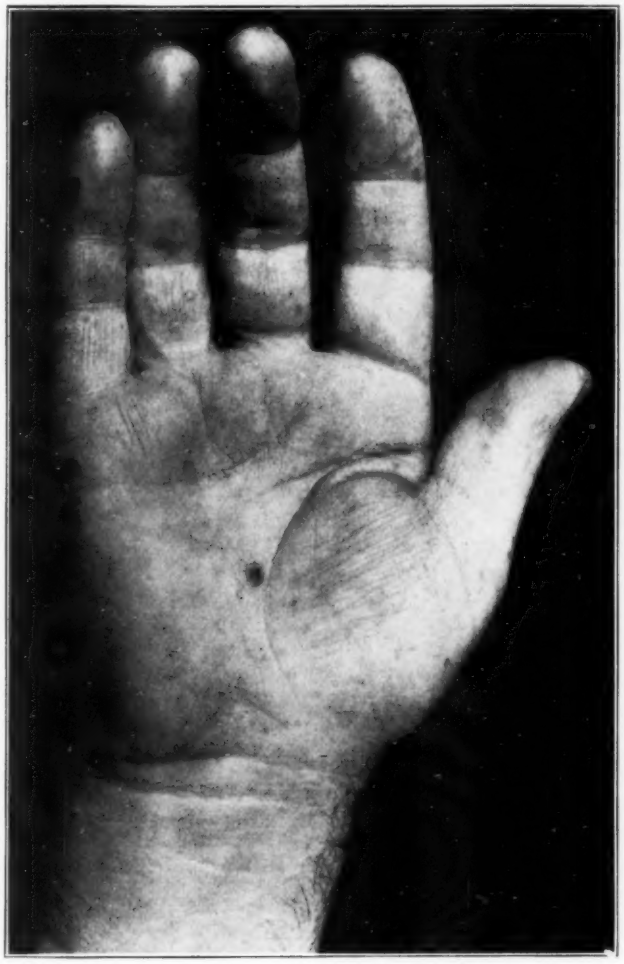

Fig. 2.—Lesions on the hand.

obtained by other authors. The lesion formed a considerably thickened part of the horny layer. No parakeratosis was seen. Within the whole section were noted two elongated sweat ducts which showed otherwise normal conditions. The stratum lucidum was thinned; the granulosum and rete malpighii were normal, except that the epithelial projections of the latter were elongated. So were the papillae of the corium beneath them. There were no signs of cellular infiltration within the papillae or within any other part of the corium, i. e., no inflammation.

ENUMERATION OF CASES REPORTED

The case reported here is added to twenty-seven as collected from the literature forming this special group. The preceding ones observed were one each by Davies-Colley,[1] Buschke and Fischer,[2] Galewsky,[3] Sweitzer,[4] Oppenheim,[5] Arzt,[6] Riehl,[7] Colomon,[8] Junghans,[9] Sklarz,[10] Rinseman,[11] Schuerch[12] and Balzer;[13] two cases each by Galloway,[14] Brann[15] and Lipschuetz;[16] and eight cases by Brauer.[17] Twenty of the cases occurred in males and seven in females. Both the palms and the soles were affected in twenty-three cases; the palms only, in three; and the soles only, in one.

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1. Davies-Colley: Disseminate Clavus of Palms of Hands and Soles of Feet, Tr. Path. Soc., London **30**:451, 1879.

2. Buschke and Fischer: Keratodermia maculosa disseminata symmetrica palmaris et plantaris, Ikonographia Dermatol., 1910, p. 183.

3. Galewsky: Keratodermia maculosa disseminata symmetrica palmaris, Arch. f. Dermat. u. Syph. **138**:445, 1922.

4. Sweitzer, S. E.: Keratoderma Punctatum, Arch. Derm. & Syph. **8**:687 (Nov.) 1923.

5. Oppenheim: Keratodermia symmetrica disseminata papulosa palmare et plantare, case demonstration, Zentralbl. f. Dermat. **6**:325 and 332, 1923.

6. Arzt: Case demonstration, Zentralbl. f. Dermat. **6**:325 and 332, 1923.

7. Riehl: Case report, Zentralbl. f. Dermat. **6**:325 and 332, 1923.

8. Colomon: Keratodermia maculosa disseminata symmetrica palmaris et plantaris, Arch. f. Dermat. u. Syph. **143**:59, 1923.

9. Junghans: Keratoma hereditarium dissipatum palmare et plantare, case demonstration, Nordwest Deutsche Dermatologische Vereingung, Nov. 27, 1921, cited by Sklarz: Arch. f. Dermat. u. Syph. **144**:297, 1923.

10. Sklarz: Keratodermia maculosa disseminata symmetrica palmaris et plantaris, Arch. f. Dermat. u. Syph. **144**:295, 1923.

11. Rinseman: Keratodermia disseminata symmetrica palmaris et plantaris, sixty-eighth meeting of the Hollandish Dermatological Association, March 30, 1924, reviewed in: Zentralbl. f. Dermat. **15**:195, 1925.

12. Schuerch: Keratodermia maculosa disseminata palmaris, Schweiz. med. Wchnschr. **55**:306 (April 2) 1925.

13. Balzer, F.: Keratome ou keratoderma punctiforme ou miliaire et de la keratodermie diffuse palmaire et plantaire avec keratomes miliaires, Ann. de dermat. et syph. **6**:225 (April) 1925.

14. Galloway, J.: Punctate Keratoses of Palms of Hands and Soles of Feet, Brit. J. Dermat. **30**:123 (July-Sept.) 1918.

15. Brann: Keratoma palmare et plantare dissipatum (Brauer), Arch. f. Dermat. u. Syph. **139**:201, 1922.

16. Lipschuetz: Cases demonstrated, Zentralbl. f. Dermat. **6**:325 and 332, 1923.

17. Brauer: Keratoma dissipatum hereditarium palmare et plantare, Arch. f. Dermat. u. Syph. **114**:211, 1913.

GENERAL CONSIDERATIONS

The distinguishing features of the disease may be summarized briefly as follows: It may begin in childhood or later, usually soon after puberty. The lesions show symmetrical distribution on the palms or the soles, or on both. The palmar surfaces of the fingers or the plantar surfaces of the toes may also be affected. Without any preceding symptoms, there appear in these regions multiple, flat, whitish to yellow, roundish, firm, horny implantations, varying in size from that of a pinhead to that of a pea and larger, within normal skin. In their smallest size they are slightly elevated and show central pointlike depressions. They may slowly increase in number and in size. In the latter event they still remain flat and become more raised, and the central depressions become larger. Otherwise there is no horny mass in the sweat pores or spontaneous desquamation on the lesions. They show no tendency to coalesce. No hyperhydrosis or subjective symptoms are complained of by the patients, except that in some cases discomfort or even pain may be felt on walking.

ETIOLOGY AND PROGNOSIS

Nothing definite has so far been disclosed in regard to the causation of this dermatosis. Oppenheim is of the opinion that it probably is due to functional disturbances of endocrine glands. But this leads us just as far as the fact that in a few cases stigmas of hysteria or neurasthenia were noted. Heredity plays, undoubtedly, a certain rôle, as in eighteen cases familial occurrence was ascertained. One generation only was affected in the two reported patients of Galloway who were cousins, and in ours whose brother also was affected; two generations in the cases of Brauer, Sweitzer, Arzt and Rinseman; and three in those of Junghans, Schuerch and Brann.

The prognosis, as regards cure, is unfavorable, as in all the cases, even those observed over a long period, no cure has been obtained.

DIAGNOSIS

The histologic observations correspond to those outlined in the description of our case. The diagnosis should be made easily from the presence of the multiple circumscribed lesions with central depressions, embedded in normal surrounding skin, their distribution being limited to the palms and the soles. The fact that the cause is unknown and that the histologic observations are typical will give further aid. Additional facts for its differentiation from similar conditions may be found in the reports of the authors cited, especially Sweitzer and Balzer. For study and comparison it will be well, however, to mention some cases which are counted as belonging to the group under discussion, but should

be separated from it; namely, those of Chalmers and Kamar,[18] Matsumoto [19] and Voerner.[20]

The case of Chalmers and Kamar had lasted eight months and showed punctate lesions and diffuse keratotic patches on the palmar surface of the hands and fingers. Subjective symptoms of itching were present after the use of water. There was exudation with crust formation on some lesions and erythema surrounding them. The patient was a woman barber who used for the care of her patrons various lotions and pomades containing gum, sandalwood oil, cloves and other ingredients. This looked rather like a case of occupational dermatosis Matsumoto's case presented circumscribed, miliary, horny efflorescences, with central desquamation and irregularly shaped patches. They were strictly confined to the palmar surfaces of the joints of the hands and fingers. In Voerner's case there were isolated minute lesions with central pits containing scales, and horny rings on the palmar and dorsal surfaces of the hands. The histologic examination showed signs of inflammation beneath the horny formations.

NOMENCLATURE

Various names have been proposed by different authors in the publication of their cases (see references) but none seems to be descriptive and appropriate enough. The ones most acceptable are those by Galloway, Sweitzer and Oppenheim. Galloway's and Sweitzer's adjective "punctate" can be accepted tentatively only, as the actual size of the lesions is that of a pinhead or larger, while the central depressions are punctate in the smallest lesions only. The adjective "papular" used by Oppenheim is correct as the lesions are circumscribed, flat elevations and remain so. Additional adjectives, as "symmetrical" and "hereditary," are superfluous, as keratoses of other groups may be symmetrical also, while heredity has not been proved in all cases of this group.

DISCUSSION

Dr. Charles J. White, Boston: I should like to know whether any scrapings were made, and whether microscopic examination was carried out on the scrapings.

Dr. James Herbert Mitchell, Chicago: I had occasion to see an interesting case of this type last year. A woman, aged 40, had punctate keratoderma on one palm. She insisted that her hand be put in condition immediately. She was satisfied with the cure. The treatment consisted entirely of roentgeno-

18. Chalmers, A. J., and Kamar, A.: Keratodermia Punctata, J. Trop. M. **20**:121 (June 1) 1917.

19. Matsumoto, S.: A New Form of Punctiform Keratodermia, J. Cutan. Dis. **36**:280 (May) 1918.

20. Voerner: Helodermia simplex et annularis, Arch. f. Dermat. u. Syph. **108**: 161, 1911.

therapy. The scrapings were examined repeatedly for fungi but none was found on any of the numerous examinations.

Dr. Charles M. Williams, New York: I wonder whether there is any connection between this and the well-known keratoderma palmaris et plantaris. If we examine every patient that comes to the dispensary we find everything from a small isolated spot to multiple lesions, and to forms covering the whole foot. I believe that many of these are variants of the same congenital anomaly, and that there has been altogether too much naming of new diseases based on minute and unimportant differences.

Dr. Lloyd W. Ketron, Baltimore: What is the difference between this and the circumscribed form of arsenical keratoses?

Dr. David Lieberthal, Chicago: Replying to Dr. White, scrapings and examination for fungi were not made.

Dr. Williams' suggestion that there may be a close relationship between these circumscribed and the diffuse palmar and plantar keratodermas sounds plausible. As in these cases of unknown etiology, there may be assumed also a certain disposition to develop horny formations in cases in which the inciting cause, as arsenic or syphilis, is known.

Although all forms of keratoderma belong to a large general class, yet it is important for scientific reasons as well as for purposes of classification to separate the various groups on the basis of pronounced differences.

A DISTINCTIVE AND SEVERE FORM OF ERYSIPELOID AMONG FISH HANDLERS

REPORT OF CLINICAL AND LABORATORY STUDIES; DEMONSTRATION OF THE BACILLUS OF SWINE ERYSIPELAS *

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The skin disease which Rosenbach [1] in 1884 designated as erysipeloid (generally known as erysipeloid of Rosenbach) is now definitely established as an infection with an organism which he isolated and which is now recognized as a human strain of the bacillus of swine erysipelas [2] (*Erysipelothrix* erysipeloids-Rosenbach [3]). This disease is one form, a mild one, of erysipeloid.

Rosenbach described the eruption as an erysipelas-like affection, frequently seen in cooks, kitchen workers, butchers and tanners, in those who handle game and fish, and in shopkeepers who handle cheese or herring, manifesting itself chiefly about the fingers and hands, and characterized by a slowly progressing, sharply defined, slightly elevated, dark violaceous, almost livid red zone which develops around the site of inoculation. The area of redness extends peripherally as the central portion fades away without desquamation. The eruption is accompanied by a sensation of burning, pricking or itching, without constitutional symptoms or involvement of the lymphatics or glands.

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* Read before the Section on Dermatology and Syphilology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

* From the Research Institute of Cutaneous Medicine, Philadelphia.

1. Rosenbach, F. J.: Mikroorganismen bei den Wundinfektionskrankheiten des Menchen, 1884; Ueber das Erysipeloid, Verhandl. d. deutsch. Gesellsch. f. Chir. **16**:75, 1887.

2. For a more detailed review of Rosenbach's reports of erysipeloid and swine erysipelas in man, of the bacillus of swine erysipelas, see Klauder, J. V.: Erysipeloid and Swine Erysipelas in Man. A Clinical and Bacteriologic Review; Swine Erysipelas in the United States, J. A. M. A. **86**:536-541 (Feb. 20) 1926.

3. Rosenbach, F. J.: Experimental Morphologische und klinische Studien der Erreger des Schweinerotlaufs Erysipeloid und Mauseseptikämie, Ztschr. f. Hyg. u. Infektionskrankh. **63**:343-371, 1909.

From the reports of others, Lehnhartz,[4] Tavel,[5] Gilchrist,[6] Lexer,[7] Ohlemann,[8] Reich,[9] it has been observed that the disease is most frequently seen from May to September. Invariably, the infection can be traced to contact (in many cases through puncture wound) with dead animal matter of divers nature, and in all but 6 of Gilchrist's 329 cases, to crab bites, or other contact with crabs. The incubation period ranges from one to five days. A distinctive feature of the erythema is its purplish red color. The disease is confined almost exclusively to the hands; it rarely progresses above the wrists, and when it does, it extends for only a short distance. Few instances of the disease occurring on other parts (soles, face and neck) have been observed by Gilchrist, Lexer and Lehnhartz.

The subjective symptoms are sensations of burning, pricking, some itching and pain (which may radiate up the arm). In exceptional cases the two symptoms last mentioned are marked. Lymphangitis and glandular involvement are rare. Suppuration has not been observed in the reported cases. Travel, alone, reports in three patients the occurrence of fever and constitutional symptoms, considerable swelling of the involved area, with pain, lymphangitis and glandular involvement. A relapse of the erythema has not infrequently been observed. The disease disappears spontaneously, in from five days to four weeks or longer without desquamation.

Subsequent observations and studies disclosed another, a severe, form of erysipeloid. It most frequently occurs in veterinarians accidentally inoculated through a prick of the needle when immunizing swine with the serum of swine erysipelas and a small quantity of a virulent culture of the bacillus of swine erysipelas (*Erysipelothrix porci*-Rosenbach;[3] *B. erysipelatos-suis*). This severe form of erysipeloid has been called swine erysipelas in man. Since Hildebrand[10] and Mayer[11] first reported the disease in 1899, an ever increasing number of cases has been reported in the German medical and veterinarian litera-

4. Lehnhartz: Erysipelas (Rose Rotlauf) und Erysipeloid, in Nothnagel: Spezielle Pathologie und Therapie 3, 1899.

5. Tavel: Das Erysipeloid, Deutsche Ztschr. f. Chir. **61**:528-544, 1901.

6. Gilchrist, T. C.: Erysipeloid with a Record of 329 Cases, of Which 323 Were Caused by Crab Bites or Lesions Produced by Crabs, J. Cutan. Dis. **22**: 507-519, 1904.

7. Lexer: Handbuch der allgemeine Chirurgie, 1903 and 1908.

8. Ohlemann, J.: Beitrag zur Kenntnis des Erysipeloids und dessen Aetiologie, inaug. dis., Göttingen, 1904.

9. Reich, M.: Ueber das Erysipeloid, Wien. klin. Wchnschr. **21**:352-357, 1908.

10. Hildebrand: Uebertragung des Schweinerotlauf auf den Menschen, Ztschr. f. Med.-Beamte, 1899, pp. 544-547.

11. Mayer: Uebertragung des Schweinerotlauf auf den Menschen, Ztschr. f. Med.-Beamte, 1899, pp. 611-612.

ture. In addition to its occurrence in veterinarians, it has been observed among laboratory workers who handled culture of the organism; among butchers, farmers, meat inspectors and cooks, in whom the infection could be traced to the handling of infected meat (pork).

This severe form of erysipeloid (swine erysipelas in man) in the majority of patients appears at a site of injury involving the fingers or hands. The incubation period of the disease in swine is three days; in man, however, it ranges from one to four days. The symptoms are those of erysipeloid of Rosenbach, already enumerated, with the exception that they are much more severe; the swelling is much greater, so that the fingers frequently become mobilized; the itching, burning and pain is considerable, the latter often preventing sleep. Lymphangitis and glandular involvement are frequently present. The disease frequently extends above the wrists, involving the forearms and arms. Constitutional symptoms and fever are uncommon. Suppuration is absent. The course of the disease is about four weeks. As evidence of a blood infection, Düttmann [12] records three cases in which the disease first appeared on one hand, then on the other hand and later on both feet. Acute fatal cases have been reported in which the picture was one of sepsis with bluish-red lesions on various parts of the body; in some, arthritis pains and evidence of endocarditis (characteristic symptoms of the disease in swine). The chronic form of the disease, as it is seen in swine, has also been observed (though rarely) in man, characterized by endocarditis and polyarthritis.

The differential diagnosis of this form of erysipeloid from the milder form is, in some cases, difficult. The following are considered in making such a diagnosis: the history of handling the culture; recent contact with hogs or the flesh of hogs; the severity of the symptoms; the greater incidence of lymphangitis and glandular involvement; the likelihood of the disease extending above the wrist.

It is the consensus of the German observers that the two aforementioned forms of erysipeloid are identical diseases differing in severity of symptoms, depending on the virulence of the organism which is intimately concerned with the source of infection, doubtless involving different strains of the organism.

ERYSIPELOID AMONG FISH HANDLERS

This report is based on a study of about one thousand cases (total number of cases observed by one of us, L. L. R) of erysipeloid among commercial fishermen working in fish pounds along the northern New Jersey sea coast, particularly in the vicinity of Seaside Park.

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12. Düttmann, Gerhard: Schweinerotlauf und Erysipeloid, Beitr. z. klin. Chir. **123**:461-469, 1921.

The disease commonly appears on the hands at the site of trauma usually incident to the bite of fish, or the prick from the scales, spines or fin of fish. The incubation period is about one day. At the site of inoculation, swelling and the characteristic purplish-red erythema appears, which progresses, involving one or more fingers, the palm or dorsum of the hand. Pain is a conspicuous symptom; it appears early; it is severe, deep seated, accompanied by constant throbbing, incapacitating and sleep preventing. Lymphangitis, enlargement and tenderness of the regional lymph nodes are common and appear early. The lymph nodes, however, do not suppurate. On deep incision, at the site of inoculation, a drop of pus may exude. Constitutional symptoms, fever, headache and malaise are invariably present during the first twenty-four hours. In some these symptoms persist for a few days or longer, at times becoming so marked as to incapacitate the patients.

The disease rarely extends from the hands to the forearms. After apparent recovery, the purplish-blue erythema may reappear at the original site or at a remote area on the finger or hand. The duration of the disease in the relatively mild cases is from two to three weeks. In these cases a relapse is not uncommon. In the more severe cases the duration is from three to six weeks. Continued use of the infected hand aggravates the disease. Mobilization of the hand expedites the disappearance of the infection.

Second attacks are common. We observed a few acute cases in which the entire arm was involved; in another patient, most of the leg was affected. These patients had a high fever and delirium, and presented other symptoms of sepsis. None of these patients presented purplish-red patches on the body as observed in the reported cases of acute swine erysipelas in man. A fatal outcome did not result in our entire series of patients. None of our patients presented arthritic symptoms such as pain in the finger joints, with or without a spindle form of swelling. Manifestations of this sort have been reported by Frank,[13] Axhausen [14] and Rahm,[15] as occurring in cases of swine erysipelas in man. Nor have we observed bulla and vesicles as part of the eruption, which at times occur in cases of swine erysipelas in man, as reported by Düttmann,[12] Veilchenblau [16] and Sieben.[17]

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13. Frank, F.: Von Erysipeloid und seiner Behandlung, Deutsche med. Wchnschr. no. 39, 1917; Zur Behandlung des Fingererysipels, Munchen med. Wchnschr. **71**:1094, 1924.

14. Axhausen, G.: Zur Diagnostik der Fingergelenkserkrankungen, Klin. Wchnschr. **2**:2197-2198 (Nov. 26) 1923.

15. Rahm, H.: Zur Schweinerotlaufarthritis der Fingergelenke, Klin. Wchnschr. **3**:224-226 (Feb. 5) 1924.

16. Veilchenblau, L.: Zur Uebertragung des Schweinerotlauf auf den Menschen, Deutsche med. Wchnschr. **47**:1030 (Sept. 1) 1921.

17. Sieben, H.: Generalisierter Schweinerotlauf, beim Menschen, Med. Klin. **21**:129-130 (Jan. 25) 1925.

It is apparent that this disease is a severe form of erysipeloid, somewhat different from that originally described by Rosenbach, the symptomology approaching that of swine erysipelas in man. From this visualization we rightly prophesied an infection due to a highly virulent strain of the bacillus of swine erysipelas which we isolated (to be discussed later). We are not acquainted with any report in the literature on erysipeloid among commercial fishermen, which concerns this rather distinctive form.

FACTORS DETERMINING INCIDENCE OF THE DISEASE

As mentioned, the disease invariably appears following the bite of fish, or the prick of scales, spines or fins; for this reason the handling of spiny fish, notable instances of which are the sea robin (*Prionotus*) and the porgy (*Chaetodipterus faber*), increases the incidence of the disease. In addition, the incidence of the disease intimately concerns contact with the slime of fish; it is greater when very slimy fish are being handled, as for example, sea bass (*Centropristes striatus*), croaker (*Micropogon undulatus*) and weakfish (*Cynoscion regalis* or *C. nebulosus*).

The season is an important factor; the disease is most prevalent during the hot months. It begins to appear in May and tends to disappear the latter part of September, conforming in this regard to the aforementioned forms of erysipeloid and to swine erysipelas.

The temperature of the water during the summer months appears to be a factor in determining the incidence of the disease. At this time, when the temperature of the water drops from the usual 68 to 70 F. to about 62 F. or lower, there is a lessened incidence of the disease [18] even though slimy fish are caught.

These fishermen come in intimate contact with many hundreds of live fish as they are removed from the nets into the boats and again from the boats into baskets. This transfer is performed mostly by hand. The exposed parts of their body and their clothes become saturated with fish slime. A uniform attitude among the fishermen toward fish slime is that it is "poison"; they anticipate the appearance of the infection if slime comes into contact with an abrasion or other epidermal defect. In all probability the bacillus of swine erysipelas which we isolated from the involved skin of some of our patients is present on the skin of the hands and clothes of fishermen, an epidermal defect or trauma doubtless serves as a means of inoculation.[19]

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18. In this regard it is to be noted that the bacillus of swine erysipelas grows best at a temperature of 68 F.

19. Arnholz (Zur Pathologie und Therapie des Erysipeloids, Arch. klin. Chir. April, 1925) emphasizes the importance of an epidermal defect in inoculation with the bacillus of swine erysipelas, causing erysipeloid and swine erysipelas in man.

Consistent with this view we observed the infection on the skin of the body at the site of trauma inflicted through the soiled fishing clothes of the patient, and also at the site of friction and presence of the soiled clothes, as for example, on the skin of the arm beneath rolled up sleeves; on the skin of the thigh, beneath the top of the boot; around the lips, from wiping the mouth with the sleeve; occasionally on the foot following a puncture wound through the boot.

It has been demonstrated that the bacillus of swine erysipelas is resistant to killing.[20] In accordance with this, we observed the infection on the feet of a vacationist, at the site of puncture wound with a fish bone [21] incurred while walking barefoot along the dried sand. We also observed patients (vacationists) infected through handling dog fish (*Squalus acanthias*), which apparently had lain on the beach for a long time and had dried thoroughly in the sun. In one patient the infection appeared at the site of a prick of a spine of a dried stuffed fish which was mounted and used for an ornament. In another patient (a cook) the infection appeared at the site of a puncture wound with a spicule of crab shell which had been boiled previously in lye water.

We observed few cases of the infection among pleasure fishermen and among the large number of vacationists who go fishing and crabbing at the seashore. Of the latter, the infection could be traced in most instances to a prick of the sea robin (*Prionotus*), a spiny fish, commonly used as bait. Such was the source of infection in two oystermen, who were never before infected, regardless of many occasions in which their

20. "The bacillus of swine erysipelas is very resistant towards harmful influences, which they probably owe to a waxlike capsule" (Schütz and Voges). Drying kills them gradually as they remain alive when subjected to a temperature of 37 C. for thirty-one days and to direct sunlight for twelve days (Sirena and Alessi). Heating to 70 C. destroys them in about five minutes. Pieces of meat not over 15 cm. thick require two and one-half hours' boiling for a complete sterilization (Stadie). Putrefaction does not destroy the bacilli in meat in four months (Stadie). Salting and pickling destroy them very slowly; pieces of meat and bacon may contain the virulent bacilli after lying in pickle for 170 days and in a mixture of salt and saltpeter for thirty days, while in smoked ham virulent bacilli were found even after three months (Petri). On the other hand, continued smoking for two weeks or shorter periods of smoking once repeated destroys the virus of erysipelas in pieces of pickled meat not over 2½ Kg. in weight (Stadie). Of the disinfectants, the following are effective: Chlorate of lime (1 per cent), hot lye (1 per cent), soda (5 per cent), iron sulphate (3 per cent), copper sulphate (0.25 per cent), corrosive sublimate (0.1 per cent), phenol, creolin and lysol (Petri), as given by Hutyra and Marek, in Pathology and Therapeutics of the Diseases of Domestic Animals, transl. by Mohler and Eichorn, Chicago, **1**:62, 1913.

21. It may be noted here that one of us (L.L.R.) observed instances of a severe local reaction resulting from penetration and lodging of a fish bone in the tonsil. Such a reaction was perhaps infection with the bacillus of swine erysipelas.

hands were cut handling oysters. In these, as in similar cases among vacationists, no infection resulted from the prior employment of menhaden or moss bunker (*Brevoortia tyrannus*) a nonspiny fish used as bait.

GEOGRAPHIC DISTRIBUTION OF THE INFECTION AMONG FISHERMEN

The commercial fishermen engaged in the fish pound industry are mostly Danes, Swedes, Norwegians, some Portuguese and Italians. Many of these men are veteran commercial fishermen who have pursued this work in different parts of the globe. They are familiar with the disease and have become fairly proficient in differentiating it from pyogenic infection of the hand. According to these men, the disease is prevalent in commercial fishermen in all parts of the world, including salmon fishermen in the Arctic Ocean. However, they say that the disease does not occur among the fishermen in the sardine industry and among the menhaden fishing crews. The menhaden (*Brevoortia tyrannus*) is a smooth, oily fish. We have noted the absence of the disease among such fishermen in the region in which our observations were made.

Our studies have been confined to the Northern Atlantic seacoast of New Jersey. From oral communication with many practitioners of medicine scattered along the New Jersey seacoast, we find that the disease is a familiar one to them, but more particularly to those physicians treating commercial fishermen. In some quarters we believe the infection is not recognized as such, but as a pyogenic infection incident to a puncture wound.[22]

We canvassed by letter many commercial fisheries that handle live fish and fish shipped to the market, also canners of fish and some physicians who treat the employees of such companies. The replies received from sources along the Atlantic coast from Maine to Florida, and from one source on the Gulf of Mexico, justify the statement that the disease is widespread among commercial fishermen, and emphasize the fact that it is an important disease of the fishing industry. Dr. Guy C. Milnor of Honolulu, H. T., informs us that the infection has been observed on tuna fishermen and on the Hawaiian fishermen who catch squid, slimy fish which wrap their tentacles around the arm of the fisherman. The infections appear on the hands and arms.

As far as we could determine, the disease is uncommon among commercial fishermen engaged in catching fresh water fish. It is to be

22. In dermatologic clinics, cases of erysipeloid are uncommon; apparently, more cases are seen in surgical clinics. It is logical to believe that there are cases of the disease treated in surgical clinics as ordinary pyogenic infection of the hand. Most of the reports on erysipeloid are from surgical clinics.

recalled, however, that this industry, in comparison with the salt water fish industry, is relatively small, being confined chiefly to catfish, black bass, crappies and bream.

ERYSIPELOID AS AN OCCUPATIONAL DISEASE [23]

In our observations the disease is the chief cause of disability among fishermen who work in fish pounds, and who handle live salt water fish. The disease is an important one of the fishing industry, not only because it is common, but also because of the period of disability, ranging from two weeks to six weeks; its reappearance after apparent recovery; the absence of immunity following an attack, and the poor response to local treatment.

In the fishing industry, indeed among some physicians, the disease is known simply as "fish poisoning."

The infection appears to be not only less frequent among handlers of fish shipped to market, both wholesale and retail, but also milder, conforming to what is conventionally known as erysipeloid of Rosenbach. In this regard it is to be noted that after fish are caught, they are partly cleansed, washed in running water, packed in ice and shipped. These measures remove slime and render the fish fairly clean and presumably less likely to cause the infection. Moreover, the wholesale and retail handlers come into much less intimate contact with the fish than do the fishermen who catch them. Again, it is possible that the organism becomes less virulent (reasons for this assumption will be discussed later).

In our canvass, the rôle that slime and puncture wounds play in causing the infection was stressed, as well as the rather long period of disability. It was of interest to note in one of our replies, "Several typical instances of the infection resulted from scratching the hand on a dilapidated ice box formerly used for the storage of meat and fish." And the following from a source concerned in the canning of fish: "It (the infection) occurs especially among the so-called bone pullers, that is, those who remove the small bones from the salt fish by means of pliers." From the same source the information was obtained that the infection occurred following puncture wound with a fish bone in the employees removing bones from haddock, which were previously steamed at a temperature of from 130 to 140 for a period of from

23. Mention may here be made of the following skin diseases common among commercial fishermen: dermatitis of the hands from handling nets treated with tar, copper sulphate or arsenical solution; an urticarial eruption alleged to be due to hemp in new nets and known to the fishermen as "hemp itch;" urticarial eruption from contact with the Portugese man of war; severe, local, sometimes systemic, reaction from a puncture wound inflicted by the spur of the stingaree, sting of the electric eel and sting of certain catfish tribes.

fifteen to twenty minutes. The more probable source of the infection in these cases is the soiled hands of the worker, the trauma of the fish bone serving as a means of inoculation, rather than the previously steamed fish bone itself.

DISSEMINATION OF THE BACILLUS OF SWINE ERYSIPELAS

The symptomatology of erysipeloid depends on the virulence of the strain.

The wide dissemination of bacillus of swine erysipelas is extraordinary. Its habitat is apparently dead matter of animal and plant origin. In view of this and considering the diet of swine, it is not surprising that, according to Glässer,[24] 50 per cent of all swine are carriers of the organism without symptoms of the infection.[25] Furthermore, the organisms were demonstrated by Prausnitz [26] in diseased cows, sheep and birds.

The reason that the infection with the swine strain of the organism in man and swine begins to appear in spring, Arnholz [19] says, is the fact that the first green fodder eaten by swine is cultivated by manure, and that for economic reasons there is a sale of the inferior meat of swine. This reason cannot be correlated with our observations relative to the factors which determine the incidence of the type of infection among fishermen.

There are three generally accepted strains of the bacillus of swine erysipelas—human, swine and mouse (*Bacillus muriscepticus*)—which have been modified in virulence and somewhat in morphology through prolonged passage in their respective hosts. It is known that the virulence of the swine strain of the organism can be modified by passage through animals and also by culture. For example, passage through rabbits renders the organism much less virulent to swine, and passage through pigeons increases virulence.

Differences in the symptomology of the mild form of erysipeloid (originally erysipeloid of Rosenbach) and of the severe form (swine erysipelas in man) can be accounted for by the differences in the virulence of the organism, depending on the source of infection. Prolonged passage in dead animal matter apparently changes its virulence.

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24. Glässer: Die Krankheiten des Schweines, 1912, quoted by Arnholz (footnote 19).

25. These figures doubtless refer to swine in Germany, where the infection among swine is more prevalent. For data relative to the infection among swine in the United States, see Klauder, J. V. (footnote 2); also Tenbroeck, Carl: Studies on Bacillus Muriscepticus, or the Rotlauf Bacillus, Isolated from Swine in the United States, J. Exper. Med. **32**:331-343, 1920.

26. Prausnitz, quoted by Pick, E.: Zur Aetiologie und Therapie des Erysipeloids, Dermat. Wchnschr. **79**:1628 (Dec. 20) 1924.

Differences in the morphology of the organism can perhaps be explained in this way.

In our culture employing a 3 year old culture of swine strain of the organism, it was nonpathogenic to white mice and to pigeons, although it is highly pathogenic to these animals. Sabella [27] observed that varying the culture mediums caused marked changes in the morphology of the organism. In Stickdorn's [28] studies in which he repeatedly cultivated the organism in agar and bouillon, the virulence in the course of one month was decreased ten times the fatal dose in white mice and to one thousand times in eighty days. The virulence was markedly decreased by repeated culture in bouillon alone. Passage through white mice decreased the virulence for these animals, whereas passage through pigeons increased the virulence for white mice, remaining unchanged for pigeons.

It is to be recalled that Rosenbach and, subsequently, others recovered the organism from patients with the mild form of erysipeloid, as coccus-like bodies, somewhat larger than staphylococci, which later grew as threads. The virulence of the organism was apparently not studied, but it is to be noted that Rosenbach and others inoculated their skin with the recovered organism and reproduced the disease.

The strain of organism we recovered from the type of erysipeloid reported herein was highly virulent, killing white mice injected subcutaneously with 0.0001 cc. The organism was too virulent for experimental inoculation in man. In view of the foregoing, it appears that the severe form of erysipeloid herein reported can be explained as an infection with a highly virulent organism, perhaps another strain—a fish strain of the bacillus of swine erysipelas. We are still engaged, however, in this phase of our study, which will be reported later. Considering the dissemination and habitat of the bacillus of swine erysipelas, it is not surprising that fish (caught three miles from shore) are apparently carriers of the organism, doubtless contaminated from refuse thrown into the ocean.

Our clinical conception of erysipeloid has changed from a somewhat arbitrary one to an infection which may be systemic, presenting a cutaneous picture of variable severity. The disease could more properly be called bacillus of swine erysipelas infection in man.

TREATMENT

The disease does not appear to be helped greatly or its course shortened by local treatment with conventional applications, such as boric

27. Sabella, A.: Involutionsformen des Bacillus erysipelatos suis, Centralbl. f. Bakteriol. **94**:411-416 (May 1) 1925.

28. Stickdorn, quoted by Kolle and Wassermann: Handbuch der Pathogenen Mikroorganismen, ed. 2, **6**:12, 1913.

acid compresses, saturated solution of magnesium sulphate, tincture of iodine, ichthyol ointment, etc. Ultraviolet ray and foreign protein therapy have been recommended, and are alleged to be of some value in treatment. We can appropriately quote the experience of others from information received in the foregoing canvass, "The condition seems more or less self limited and responds but poorly to local treatment." "Patients lose two or three weeks in spite of all that one can do." "About the only effective treatment (one questions if it really shortens the course of the disease) is rest and heat, in the form of wet packs." The last statement sums up our experience in treatment.

From reports in the German literature, the intramuscular injection of the serum of swine erysipelas, from 1 to 2 cc. per kilogram of body weight, is effective in treating the mild and severe forms of erysipeloid. Following its use, there is a rapid subsidence of the symptoms and disappearance of the lesions. A second injection is rarely necessary. This serum is universally employed in Germany in immunizing swine against the infection.

Such serum has not been available in the United States. From the strain of bacillus of swine erysipelas isolated in our studies we had prepared an immune serum.[29] This serum in doses of 0.2 cc. protected white mice against one thousand times the fatal dose. We have recently employed it clinically. Following an intramuscular injection of an amount as little as 25 cc., symptoms of the infection promptly disappeared. This serum would doubtless be of value in treatment of the milder form of infection (originally erysipeloid of Rosenbach). We have not yet employed it in such cases. We are studying in fishermen the value of prophylactic employment of vaccines.

LABORATORY STUDIES

Recovery of the Bacillus of Swine Erysipelas.—From several patients (fishermen) presenting the disease, a piece of skin and subcutaneous tissue about 3 mm. square was excised from the infected area. These specimens were placed in separate Noguchi tubes containing hormone broth and a small quantity of ground calf brain. The p_H of this medium was from 7 to 7.2. The culture was incubated at room temperature for from twenty-four to forty-eight hours, at which time smears showed considerable numbers of the bacilli of swine erysipelas coccoid in appearance and, in addition, one or the other of the following organisms: staphylococci, streptococci, a gram-negative rod of the colon group (fig. 1). Pure culture of the bacillus of swine erysipelas was obtained by inoculating white mice and culturing the heart blood, also by plating on blood hormone agar.

29. The serum is prepared by H. K. Mulford Company, Philadelphia.

Culture Medium.—The organism grew better on the hormone broth and calf brain medium than on plain hormone broth. The addition of blood to hormone agar enhanced its growth. In the broth medium a distinct clouding was produced in twenty-four hours, becoming more marked in forty-eight hours; later a sediment formed, the culture fluid becoming clear. On solid medium, the growth presented a veil-like appearance; on subcultivating the growth became abundant. This was likewise noted in broth cultures. The organism did not liquefy gelatin, nor did it ferment sugars. Cultures did not change litmus milk.

Fig. 1.—Smear of forty-eight hour culture of excised skin from patient with erysipeloid of the type herein reported, showing staphylococci, gram-negative rod and bacillus of swine erysipelas as indicated; magnified 1,200 times.

Morphology.—The organism we isolated conformed with the characteristics of the bacillus of swine erysipelas. It appeared coccoid and also as straight or slightly curved rods, from 1 to 1.5 microns long (figs. 1 and 2). It was grampositive. After cultivating for several generations, in the course of seven months, the organism assumed threadlike forms when grown on agar (fig. 3), whereas in bouillon cultures, it appeared coccoid and as rods (fig. 2).

Fig. 2.—Organism coccoid in appearance and also in straight or slightly curved rods. Smear prepared from bouillon culture after passage on culture for seven months. Carbol fuchsin stain; magnified 1,200 times.

Fig. 3.—Organism in threads when grown on agar after passage on culture for seven months; magnified 1,200 times.

Pathogenicity and Virulence.—Studies of the virulence of the organism in laboratory animals are recorded in the table. It was shown that the original skin culture injected intraperitoneally into white mice in doses ranging from 0.2 cc. to 0.001 cc. killed the animal in four days. A forty-eight hour pure culture in hormone broth killed pigeons in four days following intramuscular injections of 0.001 and 0.0002 cc.; white mice in from two to four days following subcutaneous injections of amounts varying from 0.5 cc. to 0.0001 cc. An intraperitoneal injection of 0.5 cc. killed a white mouse in two days; an intravenous injection of 0.2 cc. in a rabbit killed the animal in four days, whereas subcutaneous injection of the same dose failed to kill the animal. Subcutaneous

Results of Study of Virulence of Bacillus of Swine Erysipelas (Isolated from Patients) in Laboratory Animals

From Patient M

	Animal	Amount of Culture Injected, Cc.	Method of Injection	
Inoculated with original skin culture containing bacillus of swine erysipelas and other organisms	White mouse.....	0.2	Intraperitoneal	Dead 4 days§
	White mouse.....	0.1	Intraperitoneal	Dead 4 days§
	White mouse.....	0.01	Intraperitoneal	Dead 4 days§
	White mouse.....	0.001	Intraperitoneal	Dead 4 days§
Inoculated with pure culture (48 hour brain hormone broth) of bacillus of swine erysipelas	Pigeon.....	0.001	Intramuscular	Dead 4 days§
	Pigeon.....	0.0002*	Intramuscular	Dead 4 days§
	White mouse.....	0.5	Intraperitoneal	Dead 2 days§
	White mouse.....	0.5	Subcutaneously	Dead 3 days§
	White mouse.....	0.01†	Subcutaneously	Dead 4 days§
	White mouse.....	0.01	Subcutaneously	Dead 5 days§
	White mouse.....	0.001	Subcutaneously	Dead 4 days§
	White mouse.....	0.0001*	Subcutaneously	Dead 8 days§
	White mouse.....	0.0002	Subcutaneously	Dead 4 days§
	White mouse.....	0.0003	Subcutaneously	Dead 4 days§
	Rabbit.....	0.2	Intravenously	Dead 4 days§
	Rabbit.....	0.2	Subcutaneously	Lived
	Guinea-pig.....	0.1	Subcutaneously	Lived
	Guinea-pig.....	0.5	Subcutaneously	Lived
	Guinea-pig.....	0.2	Subcutaneously	Lived
	Field mouse.....	1.0	Subcutaneously	Lived

* Smallest amount injected.
† Animal survived injection of this amount, after passage of organism on culture, for seven months.
§ Pure culture of B. swine erysipelas obtained from heart blood.

injections of amounts varying from 0.5 to 0.1 cc. failed to kill guinea-pigs. Field mice survived subcutaneous injections of 1.0 cc. A heart blood culture of all animals succumbing to the foregoing injections yielded a pure culture of the organism.

After passage of the organism on culture for seven months, the virulence was decreased from 0.0001 cc. to 0.01 cc.

The foregoing results are characteristic of the bacillus of swine erysipelas. The organism is highly pathogenic to white mice and to pigeons and nonpathogenic to guinea-pigs and to rabbits (except in the latter when a heavy suspension is injected intravenously). The organism is nonpathogenic to field mice (a means of differentiation).[30]

30. For a further discussion of the bacillus of swine erysipelas, see Preisz, H.: Rotlauf der Schweine: Handbuch der Pathogenen Mikroorganismen, Kolle and Wassermann **6**:1-35, 1913.

The bacillus of swine erysipelas is apparently pathogenic generally throughout the animal kingdom, although virulence varies considerably in different species. Man is relatively immune to infection, particularly through the gastro-intestinal route. In human infection, the organism has an affinity for the skin.

It is of interest to note the high degree of virulence of the organism isolated; the smallest amount injected, 0.0002 cc., was fatal to pigeons, and 0.0001 cc. was fatal to white mice.[31]

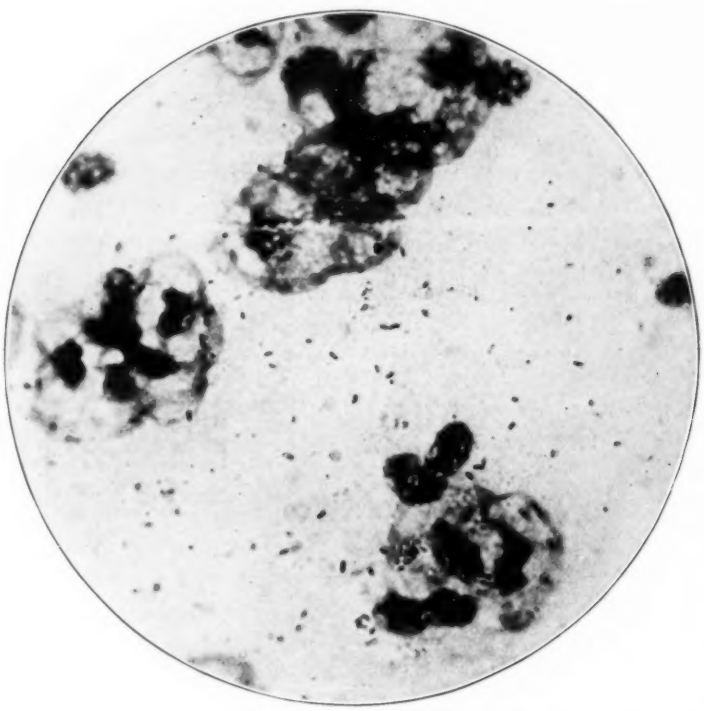

Fig. 4.—Bacillus of swine erysipelas in smear of heart blood from inoculated pigeon. Phagocytoses should be noted. Carbol fuchsin stain; magnified 1,200 times.

Studies of the virulence of the organism isolated from other patients showed a similar high degree of virulence, and for this reason are not tabulated.

Figure 4 shows the organism in a heart blood smear from a pigeon inoculated intramuscularly with 0.0005 cc. of a twenty-four hour hormone bouillon culture.

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31. Stickdorn (footnote 28) gives 0.000001 cc. of a two-day bouillon culture of the bacillus of swine erysipelas as the fatal dose in white mice.

Figure 5 shows the organism in thread form in a smear prepared from an agar culture of the bacillus of swine erysipelas furnished by Dr. John S. Buckley.[32] This strain was isolated from a diseased swine about three years before.

AGGLUTINATION AND COMPLEMENT FIXATION

The organism we isolated was agglutinated by positive swine erysipelas serum,[32] and gave positive complement-fixation reactions with

Fig. 5.—Swine strain of bacillus of swine erysipelas shown as threads. This strain was maintained for several years on culture mediums; magnified 1,200 times.

the same serum. This affords additional proof as to the identity of the organism.

We obtained a 2 plus complement fixation in the serum of one patient with an antigen of the bacillus of swine erysipelas isolated from other patients. This patient, whose serum gave a positive complement

32. This serum was obtained from Dr. John S. Buckley, chief of the pathologic division of the Bureau of Animal Industry, United States Department of Agriculture, and was that of a hog, which had received repeated injections of from 10 to 20 cc. of a heavy suspension of the bacillus of swine erysipelas.

fixation, had the infection one month before and four months before. The patients from whom we isolated the organism gave a negative complement fixation with the antigens of the organism obtained from themselves. These tests were performed eight days after their infection.

ATTEMPTS TO ISOLATE THE BACILLUS OF SWINE ERYSIPELAS FROM FISH SLIME

Through the kindness of Drs. H. S. Davis and Samuel F. Hildebrand of the United States Bureau of Fisheries, we studied slime collected and forwarded under sterile conditions from the following fish: sea robin (*Prionotus scitulus*), sea bass (*Centropristes striatus*), white perch (*Bairdiella chrysura*), pinfish (*Lagodon rhomboides*) and sheepshead (*Archusargus probatocephalus*). The slime was cultivated in hormone broth calf brain mediums. This culture and also the slime itself was injected subcutaneously into white mice. We failed in this study to demonstrate the bacillus of swine erysipelas, although we recovered various types of cocci (streptococci, staphylococci, etc.), colon bacilli and proteolytic types of bacteria.

1934 Spruce Street—1934 Spruce Street—2101 Pine Street.

RECURRENT ERYSIPELAS OF THE LEGS WITH DERMATITIS OF THE FEET*

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The preference of erysipelas for the face is well known. Dr. Joseph Doane in analyzing 1,828 cases at the University of Pennsylvania Hospital, found that 1,531, or 84 per cent, involved the face. He added Boston's and Ander's collection, which made a total of 3,092 cases, and the facial type was observed in 2,522, or 81.2 per cent. He accounts for this high frequency by the lymphatic drainage from this area, adding that the face is more easily involved, both by trauma and by bacterial infection, than other protected parts of the body. All of this is covered in detail by his paper.

Tice in "Practice of Medicine" states that 90 per cent of cases are situated on the face, and that the next most common site is on the leg, which comprises about 8 per cent. He states that it usually starts from some abrasion, or sore, but he does not go into details as to the character of the lesion on legs, nor does he refer to recurrences in this location.

LITERATURE

A perusal of literature on recurrent erysipelas would indicate that it is a rather rare condition, and the uniform localization of my cases and the rather large number, as compared with the experience of my confreres, would seem to indicate that warmer climates may be predisposing. Sixteen of seventeen cases in this paper occurred on the feet and legs, and one occurred on the hand.

REPORT OF CASES

This series consists of seventeen cases of recurrent erysipelas, but since many of them were of a common type, only those of special interest are reported in detail; sixteen were on the leg and foot, and one on the hand. The only case occurring on the hands was that of a dentist, who had a procaine dermatitis. Repeated examinations for mycelia were negative, but cutaneous tests and results proved the condition to be due to procaine, with an involvement of the ungual margin and the tips of his fingers on both hands. The recurrence of erysipelas, however, was always on the same hand, notwithstanding that the same pathologic condition existed on all fingers of both hands. His recurrences extended over a period of three years, and he had eight attacks. After the lesions on his fingers were proved to be procaine dermatitis and were

* Read before the Section on Dermatology and Syphilology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

controlled, he has not had an attack of recurrent erysipelas in about eighteen months.

The oldest patient in our series was a railroad official who had a very severe attack of erysipelas in 1914, which in addition to the symptoms above outlined showed marked prostration. His history was that he had had twenty or thirty attacks extending over a period of twelve years. He had what we now believe to be eczematoid ringworm involving both feet. It should be remembered that this was before the paper of Ormsby and Mitchell in 1916, and our diagnosis at that time was eczema. After many trials and tribulations, his feet healed, with the result that he had but one more attack and died two years ago at the age of 72 of carcinoma of the throat.

The next patient, a bank president, presented himself in 1916. Recurrences were more frequent in this case than in any in this series. He had had five attacks within a period of six months. He always ran a high temperature following a severe chill. A feature in this case is that at the present time the affected leg is much larger than the normal leg. The condition on both feet was undoubtedly eczematoid ringworm. Attention to his feet prevented recurrences of the erysipelas.

Case 3 ran a typical course, except that the swelling was not so marked as in most other cases, and while it would be ushered in with a chill, fever and reddened areas, it was very much like an ordinary streptococcic erysipelas. There would be more than one area, redness which would rapidly coalesce, and later present the characteristic features of erysipelas. In all there were six attacks.

The mildest case in the entire series, both locally and systemically, was that of a clothing salesman whose temperature never ran above 101 and the areas of involvement rarely extended above the middle third of the leg. He would usually be incapacitated for work, but did not present the appearance of a sick man, as did all the other patients.

Case 13 was interesting in that it presented an amount of systemic involvement, high temperature, etc., out of proportion to the involvement on the leg. This could only be explained by the virulence of the organism, or by lowered resistance. The patient was an office clerk and took little or no exercise.

Case 14, presented marked vesiculation extending to the knees, with characteristic symptoms of erysipelas, both local and systemic. Vesiculation was more marked than in any other case seen.

The youngest man in the series was 28, an athletic person, who is an officer of a life insurance company. He had a proved case of ringworm, and his attacks were severe. Recurrences were frequent. He was seen in the terminal period of his last attack, but his history was definitely that of epidermophytosis, and demonstration of ringworm in his feet made the diagnosis complete. This patient was seen November, 1924, and the treatment of his eczematoid ringworm resulted in permanent disappearance of the erysipelas.

TREATMENT

The treatment in these cases was unsatisfactory before the etiology of the infection in the toes was known, as there were no means of diagnosis, nor was there a treatment that was satisfactory. After the paper of Drs. Ormsby and Mitchell, it was possible, except in the case of procaine dermatitis, to find mycelia in practically every case, and the institution of treatment of the eczematoid ringworm resulted in an arrest

of the trouble. This relief continued until patients neglected their fungus infections.

My conclusions are that cases here reported are recurrent erysipelas engrafted on lesions of the feet, in which instances the streptococcus remained localized until conditions favorable to renewed activity developed. We are not prepared to say that a recurrence of the skin condition will not result in a recurrence of erysipelas, but clinically at least it would seem to be proved, first, that they are recurrent erysipelas, and, second, that the local lesions were responsible for these attacks.

The sole treatment given during attacks was symptomatic, and treatment of the local trouble was instituted after this had subsided.

DISCUSSION

Dr. William H. Mook, St. Louis: Dr. McGlasson has described the cases and discussed the causative factors so well that there is very little to say. The surprising thing is that there is not more recurrent erysipelas. In those people who have erysipelas of the face the trouble can usually be attributed to some infection in the nose. Many people pull hairs out, squeeze pimples and so on, and that frequently starts the erysipelas.

The only new information I have is something I was told by Dr. Van Studdiford and Professor Musser, both from New Orleans, who say that they are using a new treatment, and are now injecting a small amount of streptococcus serum around the periphery of the lesions in the normal skin. As you know, practically all children die if they develop erysipelas, but these physicians have been able to save several of them by this method.

Dr. Udo J. Wile, Ann Arbor, Mich.: I think the disease called to our attention by Dr. Klauder is of distinct importance in industrial medicine. I would suggest that he investigate the presence of a similar infection in the fishermen of the Great Lakes. I see each year during summer months a number of Great Lakes fishermen who present severe infections of the hands, which are evidently the result of injuries received during the hauling in of fish nets and not infrequently from the direct injury of the sharp spines in the fins of the fish which are handled by them. These infections take the form of deep-seated cellulitis, and in two cases they have led to amputation. The fishermen of the northern district are for the most part Indians, and the inclination has been to regard the infections as associated with the unhygienic conditions under which they live. In view of Dr. Klauder's work, I am inclined, however, to believe that they belong to the same group of infections which he has described for salt water fishermen.

Another point which I think might be emphasized is the use of the word "erysipeloid." Erysipelas is so definitely a streptococcus infection that I believe it would be better to restrict its use to infections of this kind, and to speak possibly of the cases which Dr. Klauder has described as "infectious erythema" or some such name.

Dr. Fred D. Weidman, Philadelphia: The point of treatment has been brought up, and the matter of injections around the lesions of erysipelas. I have had occasion to examine two or three samples of skin taken in these cases, and I wish to point out the extent to which the bacteria were found to have passed in these postmortem specimens. In one instance they had extended for 2 inches

(5 cm.) beyond the margin. There was no question as to where the margin lay clinically, for the patient had been watched before death and the margin marked. This was done because necropsy was expected. This means that the injections must extend far out if the severe as well as the mild cases are to be checked.

I wish to mention also another dermatosis which may be seen among those who handle fish. In all cases in which these persons have large numbers of furuncles which are rebellious to treatment, it should be remembered that the larvae of a certain tapeworm (*Sparganum proliferum*) may be the cause of these furuncles. This was brought out by Stiles some years ago. The specimen was found among the fishermen of the south Atlantic seaboard.

Dr. Joseph V. Klauder, Philadelphia: I am glad to learn of Dr. Wile's observations relative to an infection on the hands of fishermen on the Great Lakes. From certain sources information was received that the infection I am reporting occurs among fresh water fishermen. It is much less frequent, however, than among salt water fishermen, doubtless due to the fact that fresh water fishing on a commercial scale is considerably less than salt water fishing.

It is unfortunate that the term erysipeloid is used in describing the infection. The description of erysipeloid of Rosenbach in textbooks on dermatology, indeed in another textbook, does not adequately describe the bacillus of swine erysipelas infection in man, with its varying symptomatology. The disease could more properly be called bacillus of swine erysipelas infection in man. If the term erysipeloid is to remain, it would be desirable to designate the mild form of the infection—the original erysipeloid of Rosenbach—as erysipeloid mitis, and the severe form such as occurs following inoculation with the swine strain of the organism and also the type I report as erysipeloid ferox.

EXCRETION OF MERCURY AFTER INTRAMUSCULAR INJECTION OF MERCURIC BROMIDE, INUNCTION AND RECTAL SUPPOSITORIES*

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AND
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The efficiency of mercurial medication must depend in a great degree on the concentration of mercury in the body; this, in turn, depends on the balance between the absorption and excretion of the mercury; this, again, can be learned only through chemical analysis. The importance of such chemical studies is evident; but the difficulty of exact determination of small quantities of mercury in the excreta has naturally resulted in limiting these studies. There are only a few series of quantitative observations, each limited to a few cases, and usually confined to the urine. These are much more difficult to analyze, but they play a not inconsiderable part and, what is most serious, a variable part in the excretion. Moreover, some if not most of the analytic methods contained inherent faults, and it was difficult to judge how far these might have influenced the results. Although these results were not worthless, and may prove valuable when they have been controlled and extended, they were not sufficiently decisive to serve as a basis for rational mercury medication. It appeared advisable, therefore, to check and supplement the data, particularly along the following direction:

1. The choice or elaboration of a reliable method for the exact estimation of the small quantities of mercury that are excreted clinically.

2. The relative importance of the urinary and fecal excretion of mercury, under various conditions.

3. The relation of salivation to the excretion level of mercury. This should show whether salivation has any definite relation to the accumulation of mercury in the body.

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* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

* From the Departments of Dermatology and Syphilology and of Pharmacology of Western Reserve University, with the collaboration of Lakeside and City Hospitals.

* The expenses of this investigation have been met mainly by the Lakeside Hospital, and by a grant from the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association.

4. The comparative course of the mercury excretion for different methods of medication. The excretion should reflect the concentration of active and of stored mercury in the blood and tissues, and through these it should show the relative efficiency of these methods. The following appeared especially interesting:

(*a*) *Inunctions:* We have made some practical modifications in the application that appear successful clinically, but which should be checked by the excretion.

(*b*) *Intramuscular and Intravenous Administration:* The excretion should help to elucidate how often injections should be given and in what dosage. It should assist us in clearing up the mooted question as to the relative efficency of soluble, semisoluble and insoluble intramuscular injections.

(*c*) *Rectal Administration:* This had been suggested as a convenient and effective method, but has not been properly controlled.

(*d*) *Oral Administration:* The excretion curve should show the cause of the failure of the older routine methods, and indicate whether modification of these might be more profitable.

(*e*) *New Mercury Compounds:* The excretion of these should be compared with that of the established compounds, by similar methods.

ANALYTIC METHOD

The first part of this program, the selection of an analytic method, proved a difficult and time-consuming task. A study of the methods so far applied to the excreta showed that all, even the most accurate, fail to take into account that in dilute solutions the mercury salts are hydrolyzed and are present partly in a colloidal form, which would not be collected by the electrolytic or amalgamation methods which were commonly employed. The error may not be important under favorable conditions, but this should not be taken for granted. We thought it advisable to avoid the fault altogether and succeeded in working out a method which includes the total mercury, colloidal as well as ionic. The details of the method are being published elsewhere.[1] In principle it consists of the oxidation of the excreta by digestion with sulphuric acid and potassium permanganate, precipitation of the mercury as sulphide and enmeshment of the precipitate by gelatinous manganic hydroxide. The washed and dried precipitate is ground up with lead chromate and decomposed by heating in a glass tube at 550 C. for three hours. The volatilized metallic mercury is condensed in the cooled portion of the tube. When the entire mercury has been separated, it is collected into one globule, transferred to a calibrated capillary tube, the length of the column measured micrometrically, and transposed to the corresponding weight. According to accurate check experiments, this method permits

1. Booth, Schreiber and Zwick: J. Am. Chem. Soc. **48**:1815 (July) 1926.

the quantitative determination of 5 mg. or less of mercury in a liter of solution, and in the presence of organic matter, with a total and constant loss of 0.01 mg.

CLINICAL STUDIES

The application of this method to patients was planned along two directions:

1. The study of the urinary and fecal excretion, day by day, in individual cases. This requires the selection of suitable cases, which can be kept under close hospital supervision, with reliable collection of the daily excreta, often for several months. Even aside from the expense, this is not always a simple matter, nor is it practical for one chemist to carry the analytic work on feces and urine of more than one or two patients. The accumulation of data by this method is, therefore, a slow process; however, it is being followed as fast as possible, at present for the inunction method.

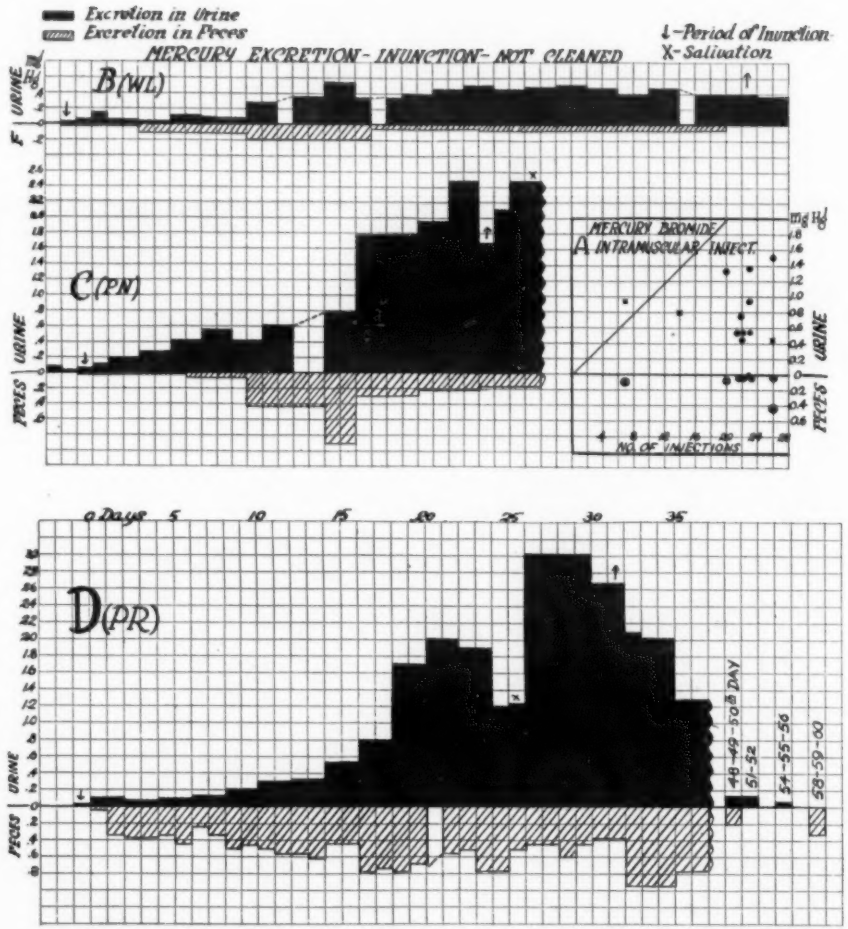
2. The analysis of three-day specimens of urine and feces of ordinary ward patients at various selected periods of treatment. These data give, in a relatively short time, a synopsis of the ordinary range of variation and of the median value. We have applied it so far only to the intramuscular injections of mercuric sodium bromide. Although our data are not yet extensive, they permit some definite conclusions, and are suggestive in other directions; and in view of the necessarily slow nature of the work, it appears justifiable to render a report of progress.

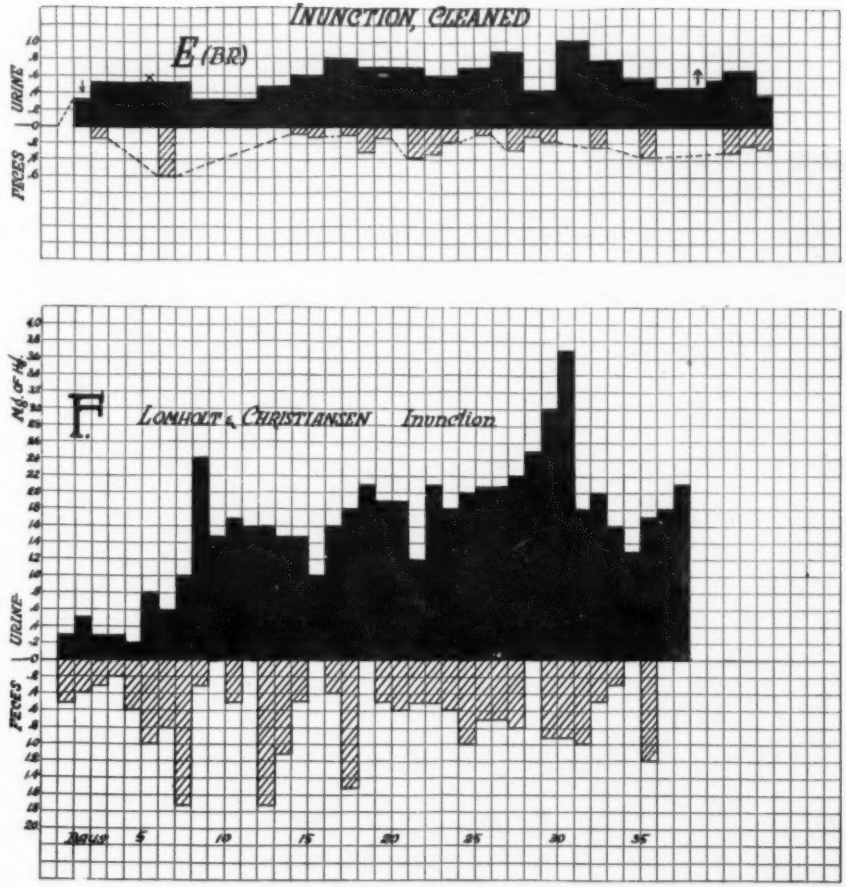
EXCRETION LEVEL WITH INJECTIONS OF MERCURIC SODIUM BROMIDE

This compound was chosen as a water-soluble mercuric salt for intramuscular injection. It is almost similar to mercuric sodium iodide, to which it was preferred because the iodide complicates the analysis. The dosage, 10 mg., equivalent to 5.5 mg. of mercury, was injected daily, in most patients for twenty-two days; in case of salivation, the injections were stopped earlier. The solution was made by the pharmacist of the Cleveland City Hospital and has the following formula:

> Four grams of sodium bromide and mercuric bromide (mercury bin bromide) were triturated together and water added to 100 cc. The preparation thus contained in the ordinary dose of 0.25 cc. (4 minims) 10 mg., equivalent to 5.5 mg. of mercury. The injections were made into the gluteal muscle. The clinical effects are indistinguishable from those of injections with mercuric sodium iodide.

The present series comprises twelve patients, the dosage ranging from seven to twenty-six injections. All the patients except CST





Excretion of Mercury by Urine and Feces.—A, after intramuscular injections of mercuric sodium bromide: The plain points and crosses above the base line represent the daily mercury excretion in the urine; the circled points and crosses represent the feces; each specimen of urine and the corresponding specimen of feces is from a different patient, who has received the number of injections denoted by its position on the horizontal line; each injection corresponds to 5.5 mg. of mercury; in the patients marked by crosses salivation occurred at that time, but not in the others; the diagonal line shows the increase of dosage. *B, C, D* and *E,* excretion during inunction (four patients); breaks in the curve, when joined by dotted lines, signify that the specimen was lost. *F,* inunction curve of Lomholt and Christiansen.

received arsphenamine, which does not interfere in the mercury determination. The details are shown in table 1, and graphically in *A* of the chart. They may be summarized as follows:

TABLE 1.—*Intramuscular Injection of Mercuric Sodium Bromide*

Patient	Age	Sex	Color	Teeth Condition	Salivation	No. of Mercury Injections	Total Mercury Injected, Mg. Hg	Daily Excretion of Mercury (Average Three Days)			Ratio: Urine / Feces	Percentage of Mercury Excreted
								Urine	Feces	Total		
M. W. S.	21	F	Black	Excellent	Mild	7	38.5	0.94	0.09	1.03	10	19
E. D. S.	30	F	White	Fair	Profuse	14	77.0	0.81	....	....	..	..
M. B. C.	20	F	White	Good	None	20	110.0	1.31	0.08	1.39	16	25
R. D. E.	15	F	White	Fair	None	22	121.0	0.49	0.01	0.50	49	9
J. G. Z.	57	M	White	Poor	None	22	121.0	0.57	....	....	..	..
R. R. R.	16	M	Black	Good	None	22	121.0	0.77	....	....	..	..
C. B. L.	25	M	Black	Good	None	22	121.0	0.58	0.07	0.65	8.2	12
F. B. C.	24	M	White	Excellent	None	23	126.5	1.37	0.03	1.40	46	25
W. W. S.	40	M	Black	Fair	None	23	126.5	0.92	....	....	..	..
C. S. T.	23	M	White	Excellent	None	23	126.5	0.47	0.03	0.50	16	9
S. S. O.	41	M	White	Fair	Mild	26	143.0	0.44	0.45	0.89	0.9	16
W. F. O.	24	M	White	Good	None	26	143.0	1.54	0.03	1.57	51	29
Median of patients with from 20 to 26 injections.....							121.0	0.58	0.03	0.89	16	16

1. The ratio of excretion bears no apparent relation to the number of injections; i. e., the daily excretion is no greater after twenty-six injections than after seven injections.

2. After from twenty to twenty-six injections, the median daily excretion by the urine is 0.58 mg. (extremes, 0.44 mg. and 1.54 mg.); by the feces, 0.03 mg. (from 0.01 to 0.45 mg.); the total excretion, 0.89 (from 0.5 to 1.57 mg.).

3. The excretion by the urine is 16 times as high as in the feces, in the median, but the proportion may be larger in the feces and ranges between 0.9 and 51 times, the fecal excretion being much the more variable. Lomholt [2] reported a narrower range, i. e., the urinary mercury was from 2 to 5 times as high as that of the feces.

4. The daily excretion represents from 9 to 29 per cent, median 16 per cent, i. e., one sixth of the amount injected; the other five-sixths accumulates in the body, at the site of injection or elsewhere, at the rate of 4.6 mg. a day, or 120 mg. of mercury by the twenty-sixth day.

5. It is important to note that this accumulation does not materially increase the elimination of mercury. This implies that the excretion rate, and therefore the concentration that is circulating actively in the blood, is not determined by the total quantity of mercury in the body.

2. Lomholt, S.: Brit. J. Dermat. & Syph. **32**:353 (Dec.) 1920. Lomholt, S., and Christiansen, J. A.: Metode til bestemmelse af smaa maengder kvaegsolv i organisk substans: Meddelelser fra Carlsberg Laboratoriet, Biochem. Ztschr. **10**, no. 3, 1913. Lomholt, S.: Arch. f. Dermat. u. Syph. **126**:1-171, 1919.

specimen of feces is from a different patient, who has received the number of injections denoted by its position on the horizontal line; each injection corre- lost. *F*, injunction curve of Lomholt and Christiansen.

THE EXCRETION CURVE FOR MERCURIAL INUNCTIONS

The excretion curve for mercurial inunctions is being determined on patients willing to cooperate, whose stay in the hospital for a sufficient period can be assured, who have good teeth and who have not recently received mercury or iodide. The general plan consists in collecting the urine and feces for from one to three days as a check, then starting the inunctions with 4 Gm. of unguentumi hydrargyri fortius U. S. P. (50 per cent), repeated daily until salivation occurs. The ointment is rubbed in by an orderly or nurse, for thirty minutes. In some cases the skin is then washed with benzene to remove the excess of ointment ("clean inunction"); in others this cleaning is omitted ("simple inunc-

TABLE 2.—*Mercurial Inunctions*

Patient	Color	Number of Inunctions	Disposal of Inunctions	Onset of Salivation	Total Daily Mercury Excretion (Mg. of Mercury) in Urine and Feces at End of 1 Week	2 Wks.	3 Wks.	4 Wks.	5 Wks.	Mercury in $\frac{\text{Urine}}{\text{Feces}}$ (Mg. and Quotients) at End of 1 Week	4 Weeks
B(WL)	White	45	Left on	None	0.2	0.6	0.5	0.5	0.5	$\frac{0.1}{0.1}=1$	$\frac{0.45}{0.05}=9$
C(PN)	White	27	Left on	30 days	0.5	1.1	2.1	2.6	...	$\frac{0.4}{0.05}=8$	$\frac{2.5}{0.2}=13$
D(PR)	White	33	Left on	27 days	0.4	0.9	2.6	3.5	...	$\frac{0.1}{0.3}=0.3$	$\frac{3.0}{0.6}=5$
E(BS)	Black	38	Removed with benzene	5 days	0.8	0.8	1.0	0.7	0.8	$\frac{0.4}{0.2}=2$	$\frac{0.45}{0.2}=2$
Median.....		36	.....	.....	0.45	0.85	1.55	1.65	...	$\frac{0.25}{0.15}=2$	$\frac{1.5}{0.4}=4$

tion"). The urine and feces are collected accurately and analyzed daily, or in convenient periods. Examinations are also made after the treatment has been stopped, when possible; but it is generally not easy to keep the patients in the hospital much longer, and it is questionable whether the collection of the excreta by ambulatory patients would be reliable. So far we have studied four patients, three with the simple and one with the clean inunctions. We are therefore unable to compare the two methods; and shall provisionally class them together. The details are shown in the chart, *B* to *E*, and the significant statistics in table 2.

F, in the chart, also shows a typical curve from Lomholt. The curves are of a similar type, but with considerable individual variations. In curves *B* and *E* the maximum excretion was obtained considerably earlier, and runs considerably lower than in curves *C* and *D* and in Lomholt's curve. The low excretion of patient *B* (W.L.) was probably

due to impaired absorption, in consequence of several generalized attacks of dermatitis repens; the last, a few months prior to our experiments with his case, had apparently subsided except for an involvement of the hands; but it is not unlikely that it had left the structure of the hair follicles impaired. The possible explanation of patient *E* (B.S.) must be deferred until there is more ample material. In the meantime, it is justifiable to consider the four cases together and to base the medians on them, as was done in table 3. Additional cases are likely to change these medians and to reveal further relations; but they are not likely to alter the provisional general conclusions, since the curves have the same direction.

1. *Course of Excretion.*—The chart and table 2 show that the excretion increases progressively with the time of administration; *B* and *E* attain a maximum in about three weeks; but the others continued to increase as long as the inunctions were continued, that is, for

TABLE 3.—*Comparison of Daily Excretion with the Different Administrations (Milligrams of Mercury; Medians and Extremes)*

Administration	Total Mercury	Mercury in Urine	Mercury in Feces	Urine / Feces
Intramuscular, mercuric bromide 2, 10 mg. 3 to 4 weeks	0.89 (0.5-1.57)	0.58 (0.44-1.54)	0.03 (0.01-0.45)	16 (0.9-59)
Inunction, 1 week	0.45 (0.2-0.8)	0.25 (0.1-0.4)	0.15 (0.05-0.3)	2 (0.3-8)
Inunction, 4 weeks	1.65 (0.5-3.5)	1.5 (0.45-3.0)	0.4 (0.05-0.6)	4 (2-13)
Suppositories 0.5 Gm. (10 day course)	.....	0.05 (0-0.09)	.....	.....

between three and five weeks. This is in marked contrast to the intramuscular injections, during which the excretion appears to remain constant after the first week, notwithstanding the progressive accumulation of mercury in the body. This means that the increased excretion that is observed in inunctions must be due to increasing absorption. This would be difficult to understand if the absorption of the mercury occurred by inhalation or through the epidermis, as has been contended by some; for there is no apparent reason why the lungs or the epidermis should absorb better at the end of the fourth week than at the end of the first week. On the other hand, it accords with the view that the mercury is absorbed from the hair follicles, for the quantity of mercury deposited there would increase with each inunction.

2. Comparing the quantities excreted with those of the intramuscular injections (table 3), it is seen that at the end of the first week the total excretion is about one-half, and at the end of the fourth week about twice, that of the intramuscular injections; the total excretion, at the end of four weeks of inunctions being 1.65 (from 0.5 to 3.5) mg. a day, as compared with 0.9 (from 0.5 to 1.6) mg. for the injections. It therefore follows that the injections would be more efficient in the first

week, but that the inunctions would overtake them before the end of the second week, and then would become materially more effective, with normal skin absorption.

3. The relative excretion by the urine and feces, the $\frac{U}{F}$ ratio, varies greatly, even in the same patient, as a glance at the statistics shows. However, it may be seen from table 3, that the fecal excretion is distinctly greater for inunctions than for the intramuscular injections, and the $\frac{U}{F}$ ratio materially lower. This point has also been noted by Lomholt, Welander [3] and others. It is also seen that the increased excretion on continued inunctions involves the urine much more than the feces, so that the $\frac{U}{F}$ quotient rises. From this it appears probable that the renal excretion responds more actively than the intestines to increase of circulating mercury. The high quotient of the intramuscular injections may be due to its less equable rate of absorption, which would give a high concentration in the blood soon after the injection; i. e., the injections would produce a daily ebb and flow of mercury concentration, the inunctions a steadily rising level.

4. After discontinuing the inunctions, the excretion declines slowly, since a store of mercury remains in the hair follicles as well as in the organs. The chart for patients, *B, C* and *E* shows that the amount of excretion remained unchanged for at least three or four days, when there was a slow decline, showing in patient *D;* but at the end of six days the excretion was still at 2 mg. Even at the end of fifty days 0.3 mg. was being excreted. The urinary excretion declines more rapidly than the fecal, according to the principle discussed in the preceding paragraph.

5. The data as to salivation are not yet sufficient for profitable discussion.

MERCURY ABSORPTION FROM RECTAL SUPPOSITORIES

Suppositories containing finely divided mercury in a cocoa butter base have been suggested as a convenient medium of administration; but their efficiency has not been established. This was attempted by administering "Mercurette" suppositories containing 0.5 Gm. of mercury, every night for ten doses. This was followed by an intermission of eight days, then another course of nine suppositories, and finally an after-period of five days. The analyses were, of course, confined to the specimens of urine. They showed extremely small quantities of mercury, between 0 and 0.09 mg., median 0.05 mg.; i. e., one thirtieth of the median daily urinary excretion of inunctions. The total quantity excreted in the urine during the two courses and the after-period was

3. Welander: Arch. f. Dermat. u. Syph. **82**:163, 1906.

0.3 and 0.5 and 0.1 mg., a total of 0.9 mg., i. e., only three fifths of the median daily urinary excretion for inunctions. This quantity was so insignificant that it was not deemed worth while to make further experiments.

CONCLUSIONS

1. The excretion of mercury from various methods of administration was studied by the method described by Booth, Schreiber and Zwick.

2. In a series of twelve patients receiving injections of a soluble mercury salt (mercury sodium bromide), in doses corresponding to 5.5 mg. of mercury, the total daily excretion at the end of from three to four weeks of daily injections averaged about 1 mg. of mercury a day. Two patients at the end of one and two weeks of injections give a similar figure.

3. Only one sixth of the injected mercury is excreted. The remainder accumulates at the rate of about 4.5 mg. of mercury a day.

4. Since the excretion does not increase with this accumulation, the excretion rate must reflect the actively circulating mercury, rather than the stored mercury.

5. With inunctions, the excretion rises progressively on continuance of the treatment, so that it is about four times higher at the end of the fourth week than at the end of the first week.

6. Since the excretion seems to reflect the actively circulating and not the stored mercury, the increasing excretion must be due to increasing rate of absorption. This would be difficult to explain on the inhalation or epidermal theories of absorption, but would be the necessary consequence of follicular absorption.

7. The rate of mercury excretion at the end of one week of inunctions averages about half that for the intramuscular injections of the soluble mercuric salt; at the end of the second week it is about the same, and by the end of the fourth week the inunction excretion is almost double that of injection. The injections are, therefore, more prompt, the inunctions presumably more powerful.

8. The ratio of fecal excretion averages higher with inunctions than with injections. Increase of absorption, however, increases the urinary excretion more than the fecal.

9. After stopping the inunctions, the excretion continues at a high level for at least a week, since the store of mercury in the follicles continues the absorption.

10. The urinary excretion of mercury during treatment with rectal suppositories of emulsified mercury indicates an insignificant absorption by this method.

DISCUSSION

Dr. Richard L. Sutton, Kansas City, Mo.: I was much interested in the observations regarding the absorption of mercury through the hair follicles. In 1905, at the suggestion of Professor Unna, I undertook some experimental work along this line, and we concluded that the metal entered the body through these portals. I am pleased that the conclusions of Dr. Cole and his associates uphold this theory.

Dr. Arthur W. Stillians, Chicago: I was interested in this paper, especially because the supposition of rectal absorption was removed. I am a little hazy on one point. If the mercury is stored so rapidly, only one-sixth being excreted after intramuscular injection, there must be an immense storage; how does the inunction method overcome this?

Dr. J. Frank Schamberg, Philadelphia: In connection with the question of excretion, one is struck, as in excretion of arsphenamine, with the minute amounts excreted through the kidneys as compared with the amount administered. The heavy metals injected into the blood are deposited in the different organs, particularly in the liver, the muscle and, at times, the bones. They are later absorbed from these organs and tissues and then excreted.

Bearing on this subject an important paper on lead poisoning has recently been published by a group from Harvard University. Lead is dominantly deposited in the bones. This is favored by the administration of calcium which is used during lead poisoning. Milk, which contains considerable calcium, is freely given. Later, when elimination becomes safe, potassium iodide or ammonium chloride is administered. It would be interesting to determine whether during mercurial poisoning the metal might not be mobilized in the bones by calcium administration and later eliminated gradually.

Dr. Harold N. Cole, Cleveland: I think Dr. Schamberg had answered the question about the storage of mercury. Miss Nora Schreiber has done the work in chemistry.

SKIN MANIFESTATIONS SEEN IN A GENERAL HOSPITAL *

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A large general hospital in which there is a friendly and close cooperation between the various departments offers to the dermatologist an opportunity to become familiar with a material quite different from the one which he generally sees in his office or even in the dermatologic wards of a hospital. The monotony of prescribing ointments or giving roentgen-ray treatment, which with some is the alpha and omega of cutaneous medicine, gives place to an appreciation that the cause of many skin diseases is found in constitutional and other systemic or metabolic factors. One learns to broaden his mental field of vision and to see in the cutaneous surface not merely an organ of protection, but an intrinsic part of the entire organism with important biologic functions. In short, one treats the patient affected with skin manifestations and not the latter alone.

We shall discuss skin conditions which we have observed over a number of years in an institution in which we are consulted in nearly every instance when a lesion or lesions appear either on admission or at any time during the patient's stay in the hospital.

A large proportion of the material consists, naturally, of drug eruptions of all kinds. As this topic was thoroughly discussed at a previous meeting of this society, we shall confine ourselves to those drug eruptions not mentioned before.

Eruptions following the ingestion of drugs are in the majority of instances readily recognized, but occasionally atypical lesions develop that require special study and differentiation. A tricky offender in this respect is phenobarbital, as illustrated by the following case.

CASE 1.—A girl, aged 15, was admitted to the hospital for a severe bullous eruption involving the tongue, lips and inner surfaces of the cheeks. The condition had been present for months, resulting in ulceration and destruction of the upper layers of the mucosa. The history revealed that the patient was epileptic and that she had been taking tablets three times daily for more than one year. As we were unable to associate such marked destructive changes with any familiar disease the question of drugs was carefully considered and the father

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* From the Service at Mount Sinai Hospital.

* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

was requested to bring the prescription calling for the tablets she had been taking. It was found that they contained phenobarbital and that she had been purchasing them by the hundred. With the discontinuance of the drug the old lesions healed, no new ones appeared and after three months the tongue healed with marked scarring and atrophy. Chemical examination of the urine revealed the presence of phenobarbital. The drug was administered a few days prior to the patient's discharge and bullous lesions again developed.

Chloral hydrate at times is followed by a severe eruption. One of the most extensive in our experience with this drug was seen in the medical ward in a patient convalescing from lobar pneumonia who developed a generalized bullous and erythematous rash with lesions in the mouth and on the lips and marked edema of the eyelids ten hours after a dose of chloral hydrate given as a sedative.

The ingestion of bromides may produce a nodular and erythematous eruption closely resembling erythema nodosum. It is generally localized on the lower extremities but is also found elsewhere and anywhere on the body, and as a rule is accompanied by a slight rise of temperature. This condition is so often encountered in our hospital that the intern makes the diagnosis, and we are called in merely to confirm it. It is surprising to us that textbooks do not mention this type of bromide eruption at all or do mention it only cursorily.

Fixed drug eruptions, to which we referred on a previous occasion as occurring after arsphenamine, phenolphthalein and antipyrine, we have also observed in a few instances since following the administration of phenobarbital and in one case of quinine.

The difficulties encountered in making a diagnosis of a drug eruption are well illustrated by the following case.

CASE 2.—A woman, aged 35, was admitted to the medical service in a comatose condition with a temperature of 103 F., and a generalized scarlatiniform eruption, congestion of the fauces and bleeding from the mouth due to erosions and ulcerations of the buccal mucous membrane. She was admitted late at night, and our opinion was sought early the next morning because the eruption was thought to be suggestive of scarlet fever or variola. In the meantime, a transfusion had been made which markedly improved her general condition. We considered two possibilities, scarlet fever and a toxic or medicamentous erythema, inclining more to the latter. It was impossible to arouse her from her stupor to obtain an intelligent answer regarding drugs, but we were disposed to believe that mercury or arsenic had been taken, probably with suicidal intent. Subsequently, the patient showed hematuria due to hemorrhagic lesions in the bladder, with a marked impairment of renal function which threatened her life for a few days. The skin manifestations gradually cleared up, and she left the hospital in a fair condition after a few weeks. A chemical examination of the urine had shown large quantities of arsenic. The patient steadfastly denied ever having taken any drug.

In the surgical wards of the hospital where acute cases are admitted for observation and operation the dermatologist is not often consulted.

Occasionally, however, postoperative cases develop acute skin conditions requiring a differential diagnosis. A superficial cellulitis associated with an injury or surgical wound often resembles erysipelas, and it is most important to make a correct diagnosis on account of the protection of other patients.

Again, localized or generalized erythematous eruptions may follow the use of iodoform as a powder or packing, or the introduction of boric acid powder into a thoracotomy wound, as we have observed in one case. If the cause is not recognized the condition may become aggravated and interfere with the convalescence of the patient. Several times we have found an iodoform gauze packing to be the etiologic factor in a case which, on account of its appearance and the accompanying rise of temperature, simulated erysipelas.

The dermatologist, in spite of many years of clinical training, will find difficulty in recognizing certain skin manifestations associated with or complicated by some of the infectious diseases seen in a general hospital. Often the eruption is one of a chain of symptoms which confirm the diagnosis. For instance, we are not infrequently asked to express an opinion whether certain skin lesions designate typhus or typhoid fever before laboratory tests have been completed. A differentiation is not always easy but, as a rule, in typhus we find hemorrhagic macules usually on the flanks and flexor surfaces of the arms, while in typhoid fever there is an erythematous and papular eruption which is more marked on the abdomen.

Not infrequently we are made the arbiters, especially in the admitting service, in the differential diagnosis between scarlet fever and measles and toxic or medicamentous erythemas. An early and definite diagnosis is of supreme importance in the protection of other patients. At times even the most experienced will make an error. This was illustrated in the following case.

Case 3.—A girl, aged 14, admitted to the hospital for an ulceration of the tongue, was given 0.25 mg. of tuberculin subcutaneously in order to exclude tuberculosis. She developed, after seventy-two hours, a severe febrile reaction, the temperature rising to 103.4 F., and a scarlatiniform eruption, with a sore throat. As we had never seen this type of eruption following tuberculin, we called into consultation one of our colleagues who specialized in contageous skin diseases. He pronounced the case scarlet fever. The patient was removed to an isolation hospital where it was said the disease ran a classic course. About two years later, the same patient was readmitted to the hospital with a large ulceration occupying the former site. We recalled the scarlet fever episode and still being of the opinion that the lesion was tuberculous, although the first injection of tuberculin did not cause any focal reaction, we again ordered a tuberculin test. Almost to the minute, that is, seventy-two hours later, the patient developed a generalized eruption, this time morbilliform with a rise of temperature to 104.5 F., which disappeared within a short time. This repetition could be interpreted in only one way, namely, that the first rash was not scarlet fever

but an unusual belated reaction to tuberculin. The final diagnosis of the lingual ulceration was hysterical mutilation, admission of self infliction being gained from the patient later.

Another experience of interest occurred about one year ago when we were called at midnight to see a very sick man in the reception ward.

CASE 4.—A man, about 30 years of age, was covered with a bullous hemorrhagic eruption. The intern on the service suspected variola. The patient also had confluent, tense vesicular lesions on the palms and soles, ulcerated lesions on the lips, tongue and inner surfaces of the cheeks, a marked angina and a high temperature. He had been ill only a few days. It was difficult to make an absolute diagnosis at the time. The patient, however, was isolated, and within twelve hours he developed on the trunk and hands typical lesions of erythema multiforme.

The medical wards have offered us a large field for the study of purpuric eruptions because of the opportunity we had of following the work of Dr. E. Libman in subacute bacterial, rheumatic and other forms of endocarditis, as well as that of the late Dr. N. E. Brill in thrombopenic purpura. The former has familiarized us with a special form of valvular and mural endocarditis which is accompanied by petechiae in the conjunctivae and various parts of the skin, generally occurring in crops and many having white centers. Again, we find an erythematous eruption of butterfly distribution closely resembling lupus erythematosus acutis, which is sometimes accompanied by erythematous lesions in other parts of the body and ulcerative lesions of the buccal and lingual mucosa.

These cases bear a certain resemblance to the erythema group of Osler. In a few instances we have seen generalized erythematous eruptions accompanied by intestinal crises which simulated acute appendicitis. In two cases in which appendectomy had been performed, the appendix was found to be normal, and the crises were not relieved.

Cases of purpura presented themselves of the type of simple or rheumatic purpura and the purpura hemorrhagica or chronic thrombopenic purpura. To differentiate between these two conditions, so different in their prognosis, was often difficult, if not impossible, from the skin manifestations alone. In the former the lesions are polymorphous, not simply hemorrhages, but combinations of hemorrhages with erythema, urticaria or edema, producing a picture which is diagnosed as erythema hemorrhagicum, while in the graver form we have simply hemorrhages of the skin and mucous membranes; but the final diagnosis can be made only by an examination of the blood (thrombopenia, bleeding and coagulation time, etc.).

A peculiar combination was observed in a young girl who had been suffering from a severe thrombopenic purpura, of which she was cured by a splenectomy. After her discharge from the hospital, a chronic lupus

erythematosus disseminatus (not the acute type) developed which in the course of about six months cleared up entirely without any medication.

In a few cases of polycythemia we saw rosacea as an accompanying symptom. In one, a generalized, obstinate, prurigo-like urticarial eruption, which we designated urticaria necrotica perstans, was observed. With the improvement of the underlying condition, the skin manifestations cleared up spontaneously.

Erythema nodosum and erythema multiforme types of eruption are frequently encountered in patients suffering from acute rheumatic conditions. We have also seen phlebitis and thrombophlebitis migrans associated with painful irregular nodular lesions simulating erythema nodosum. There is usually a rise in temperature and severe pain in the involved area. In a case reported by Dr. Harkavy, partial thrombosis of the hepatic veins, with ascites and painful ulcerations of the buccal mucosa, was observed.

Rheumatic endocarditis is not uncommon at all ages. In those suffering from acute exacerbations with a slight temperature there are occasionally seen on the trunk, and especially on the abdomen around the umbilicus, pale red, macular crescentic or semilunar lesions known as erythema annulare (Leiner). As a rule, there are no subjective symptoms, and the cutaneous manifestations are found only during the physical examination of the patient. We have seen two cases of rheumatic erythema annulare, one in a child, through the courtesy of Dr. Schick, and the other in an adult in the medical service.

Multiple telangiectasias are not infrequently accidental observations. When seen on the palms, as is frequently the case, they appear at first glance as erythematous areas; careful examination, however, discloses the minute blood vessels that make up the lesion. Our first series of cases were mostly in women suffering from visceral syphilis. Later we observed the same condition in a few patients with exophthalmic goiter. We believe the lesions are due to a disturbance of the autonomic nervous system; there is either a disturbance of the vasomotor center or a peripheral disturbance of the sympathetic system caused by pressure. Their preponderance in women is probably not accidental, as women more frequently suffer from an unbalanced autonomic nervous system than men.

In contradistinction to these cases of telangiectasia, we have seen in the medical wards a few instances of the Osler type of telangiectasia hereditaria hemorrhagica with bleeding from the mouth and nose. In one of these cases thirty-two members of one family, male and female, suffered from this condition.

Nevus anemicus and leukoderma acquisitum centrifugum were seen as accidental observations, as well as leukoderma associated with parapsoriasis, pityriasis rosea and pityriasis versicolor.

In a number of cases of splenomegaly we found marked pigmentation of the skin in scattered areas, as well as pustular sweat duct lesions on the lower extremities, which if not pathognomonic are suggestive, as the late Dr. Brill and Dr. Mandelbaum have stated in their classic paper on Gaucher's splenomegaly.

A few years ago New York experienced an epidemic of encephalitis; at that time the neurologic wards of the hospital were filled almost to capacity with patients having this disease. It was noted that a large percentage of these patients had a marked seborrhea of the face and chest with a profuse oily secretion covering these areas. The condition was so striking that the neurologists considered it one of the symptoms in favor of the diagnosis in doubtful cases.

During this epidemic we were called in consultation to diagnose the eruption in the following case.

CASE 5.—A man, aged 40, in a semistuporous condition, had an oculomotor palsy and a low grade temperature, and gave a history of headaches and malaise for about two weeks prior to admission. Examination revealed a late secondary syphilitic eruption of the extremities and palms, and moist papular lesions around the scrotum and anus. The patient's blood and spinal fluid were strongly positive, the latter showing a high cell count.

Because of the epidemic of encephalitis at the time one can readily understand how this disease would occupy the foreground and a syphilitic meningitis be overlooked.

It is well known that many aggravated and extensive skin conditions improve rapidly when the patient is placed in a hospital. The pediatrician is especially impressed by this fact in controlling infantile eczemas and papular urticarias due to food sensitization or some factor of unknown origin. Such children show prompt improvement with the same local therapeutic measures that have been employed by the parent or nurse at home. Why hospitalization should have this marked benefit on skin manifestations is inexplicable. Often these obstinate eczemas and urticarias reappear shortly after the child returns home.

In the children's pavilion the dermatologist occasionally sees unusual skin lesions as symptoms of a constitutional disease, such as lichen scrofulosorum and disseminated lupus lesions with tuberculous meningitis.

Mention should also be made of the occurrence of various types of urticarial eruptions and prurigo-like lesions in patients suffering from nephrosis. Under treatment, and with the improvement in the underlying condition, the eruption becomes milder and the itching less intense, and involution takes place.

Dr. Schick permits us to report an interesting case which we saw on account of a skin eruption.

CASE 6.—S. R., a boy, aged 2 years, was admitted to the hospital with a history of temperature of three weeks' duration. There was no cough, coryza or cutaneous eruption; no vomiting or diarrhea; no melena or hemoptysis. The child had an evening temperature of 102.5 F., and complained of pain in the abdomen. The Schick, Mantoux and Wassermann tests all gave negative reactions; the spinal fluid and blood cultures were likewise normal.

A roentgenogram of the chest showed a mottling of the lungs resembling miliary tuberculosis and an enlargement of the heart on the right and on the left. A roentgenogram of the bones showed a rarefaction of the femora and ilia, and small areas of destruction in the fourth, eighth and tenth ribs, the bodies of the lumbar vertebrae and the skull. The entire picture suggested metastatic malignancy. Six weeks after admission skin lesions developed on the hairy border of the frontal region of the scalp, throughout the scalp, and on the neck, chest and upper extremities. They were papular and pustular, varying in size from that of a pinhead to that of a split pea, resembling morphologically acne varioliformis. Their superficial character militated against the diagnosis of a tuberculid. Later the lesions became macular and erythematous and some purpuric. At this time, a marked enlargement of the cervical glands was also noted. Blood cultures continued to give negative reactions. A differential count showed the following: white blood cells, 11,300; polymorphonuclear leukocytes, 69 per cent; lymphocytes, 25 per cent; mononuclears, 5 per cent; myelocytes, 1 per cent; red blood cells, 3,900,000; hemoglobin, 50 per cent.

On October 24, soft tumor masses were noted under the scalp and right temporal region. Aspiration of one of these showed large endothelial cells. The diagnosis of endothelioma was considered. The child died several weeks later, and at necropsy disseminated multiple endotheliomyelomas were found.

Since 1915 we have seen in our service eleven cases of acute lupus erythematosus, all of them fatal. The female sex predominated in the proportion of 10 to 1, and the ages varied from 18 to 38 years. Two of the women were pregnant; in one the eruption disappeared immediately after confinement, but recurred shortly after; in the other artificial labor resulted in a marked but unfortunately only temporary improvement.

A few patients with acute lupus erythematosus were admitted to the medical wards with symptoms of bronchopneumonia. The skin manifestations they presented may be said to be pathognomonic. The face, chest and extensor surfaces of the forearms were covered with an erythematous, sometimes scaly, eruption; the palms and fingers were involved with an erythematous and telangiectatic rash, the latter involvement often confirming the diagnosis. These patients usually run a septic temperature for weeks, ranging from 101 in the morning, to 103 or 104 F. in the evening, and finally die of a septic pneumonia.

We have occasion to observe a large number of conditions of the mucous surfaces of the oral cavity, which in our institution are considered as lying in the domain of the dermatologist. Lingua scrotalis

and lingua geographica are commonly seen in the medical wards and excite little discussion except among those interns who are not familiar with the conditions and look on them as syphilitic lesions.

A more interesting and important condition of the tongue, identical with or resembling the so-called Moeller's glossitis, occurs mostly in women with pernicious anemia in which the tongue condition together with an achylia gastrica is often the earliest and the only symptom.

It is no coincidence that five of the six cases published by Moeller in 1851 were observed in women suffering from *Bothriocephalus latus,* which, as we know, causes anemia. The symptoms of this condition vary greatly. The obective picture is one of bright red irregular patches on the dorsum of the tongue, or linear lesions on the tip and sides, either with prominent papillae or smooth and atrophic, associated with intense pain and burning. We do not wish to give the impression that in all cases of Moeller's glossitis pernicious anemia is the underlying cause or that every case of the latter condition is combined with a glossitis. It is our impression that in descriptions of and discussions on Moeller's glossitis too much adherence to the original article of Moeller, published seventy-five years ago, is indulged in. Greater emphasis should be laid on the combination of these symptoms, i. e., painful glossitis, achylia gastrica and a characteristic or suggestive blood picture.

The following was an unusual case of agranulocytic angina with skin lesions.

CASE 7.—A woman, aged 33, five months before admission to the hospital, had had a mild attack of scarlet fever and had developed a sloughing sore throat perforating both anterior faucial pillars, which was followed by pulmonary suppuration. She expectorated large quantities of foul smelling pus. She was admitted to the hospital with a marked stomatitis, chills and fever of two weeks' duration, and pain on defecation. When we saw her twenty-four hours later, there was a gangrenous lesion on the inner surface of the lower lip, eroded lesions on the soft and hard palate and an eruption of the body, more marked on the buttocks, consisting of bright red, round and oval papular lesions with yellowish centers. We were in doubt whether this eruption was due to a drug or embolic in nature. It was only after an examination of the blood by Dr. Nathan Rosenthal that the diagnosis was made. The lesions in the mouth showed no Vincent's organism. The blood culture contained *Streptococcus hemolyticus* and *Bacillus coli communis* on two occasions. The blood picture, and this is the essential point, showed normal hemoglobin and a normal red cell count, but a low white cell count—only 600 cells, with 95 per cent lymphocytes and 5 per cent monocytes. This blood picture with an exceedingly low white cell count and the absence of granular cells is characteristic of agranulocytic angina. The patient died three days after admission.[1]

Our interest in this case lay in the combination of this condition, the exact nature of which is still unknown, with an eruption of the skin such as we have seen in other cases of bacteremia.

1. This case will be reported by our medical colleagues with complete postmortem observations.

In the surgical service we had the opportunity of observing an interesting case in which the skin manifestations enabled us to make a surgical diagnosis.

CASE 8.—A boy, aged 10, mentally deficient since childhood, had convulsive seizures about once every six or seven weeks. He was taken sick about one month before admission complaining of weakness, lassitude and loss of appetite. For ten days he had been having pains in the right loin, and he had had a temperature as high as 104 F. for the last three days. There were no bladder symptoms.

We were consulted on account of an eruption on the cheeks, nose and chin. The eruption consisted of numerous pinhead to large pea sized nodules, yellowish to dark red, symmetrically distributed over the involved areas—a typical example of adenoma sebaceum. Noticing the dull apathetic expression of the boy, without knowing the history of the case, we inquired whether the patient had a kidney tumor. We were aware of the fact that adenoma sebaceum with imbecility and epileptic seizures and tumors of the kidney constitute a clinical entity known as tuberose sclerosis. Operation was performed subsequently and a hypernephroma of the right kidney removed.[2]

In our institution the Wassermann test is a routine measure. We have repeatedly expressed the opinion that this test is a good servant but a bad master. It signifies by no means that the symptoms of the patient, whatever they may be, are due to syphilis. Frequently we are asked to suggest treatment in patients with a severe anemia, enlarged liver or spleen, intrathoracic or intra-abdominal tumors ,with a positive Wassermann reaction.

The following case is mentioned to illustrate that patients with a strongly positive Wassermann reaction may also suffer from totally unrelated diseases.

CASE 9.—A Chinaman, aged 40, was admitted to the medical ward for a severe jaundice of two months' standing. Physical examination disclosed a large liver, slightly irregular in outline, not very tender. Reaction to a routine Wassermann test was reported strongly positive with all methods. This brought up the question of the procedure to be followed. We suggested intramuscular injections of bismuth and small doses of neo-arsphenamine. These were administered without improvement in the jaundice or general condition. The patient was unable to retain solid food, and even liquids were expelled. After a stay of two months in the hospital, he died. At necropsy an infiltrating carcinoma of the liver, involving especially the region of the gallbladder, was found.

On the other hand, we recently had the privilege of observing three patients who had syphilis of the stomach with a severe anemia resembling primary (pernicious) anemia. One of the cases offered such interesting features that we report it in detail.

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2. This case has been reported in detail by Dr. A. Hyman of the surgical staff of the hospital, J. Urol. **8**:317 (Oct.) 1922.

Case 10.—A. R., aged 43, Russian by birth, was admitted to the medical ward in 1919, complaining of nausea, epigastric pain and paresthesias. He had a smooth atrophy of the tongue and a strongly positive Wassermann reaction. In the course of examination a periostitis of the right tibia was found. A roentgen-ray examination of the gastro-intestinal tract gave essentially negative results; gastric acidity was somewhat reduced. Microscopic examination of the blood, which was strongly suggestive of pernicious anemia, showed: color index, 1.2; megaloblasts, macrocytes and a leukopenia. The hemoglobin content was 65 per cent on admission, dropped to 25 per cent one month later, and rose to 40 per cent on discharge, following a transfusion of 500 cc. of blood. He was given arsphenamine and injections of mercury while in the hospital and advised to continue treatment at the outpatient department on his discharge.

He improved sufficiently to return to work until the early part of the summer, 1925, when he again complained of epigastric pain. This time there were no paresthesias. Examination of the blood and spinal fluid for syphilis gave negative results, but after a few injections of mercury and iodides by mouth the blood test became + +. He also showed a profound anemia with a color index as low at times as 0.5; there were no macrocytes, but a relative polynucleosis with slight leukopenia. His stomach showed a large filling defect in the fundus; there was no free hydrochloric acid in the Reyfus test; the total acidity was 40. The surgeons called into consultation pronounced that operation could not be performed. Under continued mercury, iodides and roentgen-ray treatment he improved clinically and radioscopically, and was discharged on Oct. 14, 1925.

On November 25, he was readmitted because the roentgenogram showed a recurrence of the lesion. He again had a secondary anemia, the blood picture suggesting to Dr. Rosenthal, our blood expert, a malignant neoplasm. There was no palpable mass in the abdomen. In spite of a hemoglobin content at 35 per cent, the patient was not markedly asthenic. As he reacted badly to arsphenamine, he was given bismuth intramuscularly. After two months there was no roentgenographic evidence of a lesion of the stomach, and with the internal administration of dilute hydrochloric acid three times daily the gastric symptoms disappeared. He was discharged, Feb. 1, 1926, with a hemoglobin content of 48 per cent.

We have observed lymphoblastomas in the medical wards. In these cases there is usually a progressive enlargement of the lymphoid tissues. These cases are usually complicated with urticaria, prurigo lymphatica, pruritus and erythrodermia; occasionally we saw lymphatic infiltrations but hardly ever real lymphatic tumors.

DISCUSSION

Dr. Charles M. Williams, New York: The first thought that comes to mind is the connection between the general hospital and the skin clinic. Most of us who do no work in general hospitals do not see these cases, and yet they are important in dermatology.

Another point is the difficulty of getting a history of taking a drug in the cases of suspected drug eruption. This is a familiar fact. Last week a patient came in who had a condition that looked like a drug eruption, but he said that he had not taken any drug. Later, he confessed to one of my assistants that he had taken Ex-Lax for years and some acetanilid. Anything that can be

obtained in dry form from the druggist, that is not prescribed by a physician, is not considered as medicine, at least by most of these patients.

I was much interested in the reported cases of hereditary telangiectasia and nose bleed. These thirty-two cases in one family, I think, form the largest group on record, but there was another family reported several years ago about as large. One has the impression that this is rather rare. It is not mentioned in textbooks, but if you ask every patient with spider nevus if he or some member of his family has nose bleed you will get a positive answer in a majority of the cases. In the last few weeks I have collected about eight groups giving a typical history of familial telangiectasia and nose bleed. These cases are not found except by careful questioning. I would like to know whether Dr. Rosen has had any experience in this connection.

Dr. William Allen Pusey, Chicago: The prime business of most of us is clinical medicine, and no one can listen to a paper of this sort without arousing some reactions from his experience. It is a bird's eye summary, but this makes the summary no less important.

Dr. Herman Goldenberg, New York: Dr. Williams' question regarding the combination of spider nevus and nose bleed I cannot answer; I am not familiar with it.

As to erythema being due to kidney stones, we can exclude it, at least in two cases we have reported, since a careful abdominal examination was made. Whether or not they occur in other cases I do not know.

There are, I am sure, many general hospitals in America which have excellent material, but I am equally sure there is no other hospital in which there is such a close relationship between the dermatologic and the other departments. One reason is that my associates and I always cheerfully respond to any call made on us. We discuss the cases with the attending and house staff, and I, at least, and I think I can say the same thing for my associates, derive more knowledge from cases seen in general wards than from those in our own ward for diseases of the skin. It gives us a broader view of the relation of skin diseases to general medicine and is a stimulus for us to keep up our interest in the latter.

POINT-GAP SPARKOVER VARIATIONS AND THEIR EFFECT ON DOSAGE IN SUPERFICIAL ROENTGEN-RAY THERAPY *

A. J. MARKLEY, M.D.

DENVER

The indirect or mathematical method of measurement of superficial roentgen-ray dosage as described and introduced by MacKee,[1] and Remer and Witherbee [2] has for some years held a position of primary importance in dermatologic therapy. While a more scientific method may be desirable and will no doubt be developed, this one, because of the readiness with which it may be adapted to the conditions of office practice, has fulfilled a genuine requirement and has made possible the employment of practical roentgen-ray therapy by many to whom it would otherwise have been denied.

In order that such a method may be universally applicable or in modern terms be "standardized," it is requisite that the formulas by means of which it is applied be composed of factors which remain constant in value under all conditions. It is now recognized, however, that in the formulas originally devised for this method and still largely used, there are certain inaccuracies which lead to considerable discrepancies of dosage when the method is applied under conditions different from those under which it was developed. One of the most important of such inaccuracies, and the only one here considered, is the use as a factor in these formulas of a point-gap sparkover of fixed length, instead of and as equivalent to the voltage passing through the tube. This is an inaccuracy because the voltage value of this factor is an inconstant quantity. The length of a point-gap sparkover induced by a high tension current is determined not simply by the voltage of that current, but also by certain other conditions. Influenced by these conditions, a point to point sparkover of fixed length may be induced by currents of widely varying voltage and conversely a point to point sparkover of widely varying length by a current of fixed voltage. The sparkover voltage relation is therefore irregularly variable instead of fixed and definite.

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* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

1. MacKee, G. M.: Arithmetical Computation of Roentgen Dosage, J. Cutan. Dis. **37**:783 (Dec.) 1919.

2. Witherbee, W. D., and Remer, John: A Practical Method of Roentgen-Ray Dosage Without Aid of the Radiometer, Arch. Dermat. & Syph. **1**:558 (May) 1920.

The more important of these conditions and the manner in which they affect particularly the point to point sparkover may be briefly outlined as follows.

THE CHARACTER OF THE POTENTIAL

The wave shape, length, frequency and uniformity of the potential are features that may vary with the design and construction of the apparatus used.

Because of their peculiar wave shape and length, coils produce a longer sparkover for the same voltage than do interruptless transformers; confusion from this source, however, has largely disappeared since coils have been generally superseded by transformers.

With the tendency to the use of higher voltages, wave frequency and oscillation are sources of irregularity in point-gap sparkover for which reason 50 kilovolts is about the upper limit of accuracy of measurement by the point-gap.

PHYSICAL STATE OF THE AIR

The density, humidity and temperature of the air through which the spark travels are of definite importance in determining the length of point to point sparkover.

Air Density.—Air under pressure is a poor conductor of electricity, but with the decrease in air pressure and density incident to increase in altitude the conductivity of the air increases until at great elevations the length of sparkover for a given voltage may be several times that of the same voltage at sea level.

At voltages below 50 kilovolts the point-gap sparkover for a fixed voltage decreases or increases in length in definite ratio to changes in air density; at higher voltages this regularity is lost because of the influence of other conditions.

Humidity.—This is one of the most puzzling and uncertain of the conditions that influence point to point sparkover. It is not possible to separate its effects from those of other conditions with which it is always associated. High humidity decreases, and low humidity increases, the length of point to point sparkover, probably because of the effects of corona in condensing and collecting moisture about the terminals and thus enlarging them and changing their shape.

During the winter months of close, warm, dry offices, humidity occasions little trouble, but in summer, with open windows, outside conditions effect rather startling alterations in the spark-gap.

Temperature.—Temperature influences the length of point-gap sparkover in exact proportion to its effect on air density, which under ordinary conditions is unimportant.

CORONA

Corona or brush discharge is a dominant factor in determining the length of point to point sparkover. It is the first evidence of dielectric stress, appears at every sharp point in a dielectric field, and increases in intensity with increasing voltage. It appears at voltages much lower than that of breakover, but it ionizes the air, increases its conductivity and permits breakover at a voltage lower than normal. Corona becomes, of course, more evident in the lowered air density of higher altitudes and, in conjunction with changes in humidity, causes not only variations in length, but also great irregularities in point to point sparkover.

VARIATIONS IN THE SHAPE AND SIZE OF THE TERMINALS

Variation in the shape and size of the terminals between which the spark passes is a frequent cause of sparkover variations and is generally well known. The sharper the terminal points the more concentrated is the electric tension and the more readily breakover occurs; the more nearly the terminals approach spherical in shape and the greater their size, the less is the electric tension and the shorter the sparkover until with large spheres the sparkover of high voltages may be a few millimeters.

THE PERSONAL EQUATION

The personal equation of the operator enters definitely into the proper estimation of voltage by the point spark-gap. The rapidity with which the gap is closed, the promptness of stopping the moving point at the instant of breakover, the amount and duration of corona that is permitted, the distinction between line-surge or circuit-closing breakover and that of the actual voltage—all these factors are important and make the accurate use of the point spark-gap much less simple than it really seems.

Up to 50 kilovolts (about a 3 inch [7.6 cm.] gap) the point spark-gap is a fairly accurate estimate of voltage, but with higher voltages, it becomes increasingly inaccurate because of the intricate and complex effects of the conditions just discussed, and a few illustrations will indicate more clearly the extent which these inaccuracies may reach.

It is understood that the point to point sparkover distances given are only approximate since it is not possible, because of the complex origin and erratic behavior of this phenomenon, to plot charts and curves for it, as is accurately done for the sphere-gap. Also, in each plate the variations shown are attributed to alterations in a single one of the conditions influencing the length of sparkover. This, of course, never occurs since alteration in one such condition is always associated with, or dependent on, alterations in others; actually, therefore, sparkover variations are in general greater than those shown (figs. 1, 2, 3 and 4).

It has been fully proved that the formula 6 inch (15 cm.) spark-gap, 2 milliamperes of current 3 minute time, 8 inch (20 cm.) distance, used with modern apparatus and careful manipulation, will produce the skin unit amount of roentgen-ray; and few operators think it necessary to check their use of it by tests of any kind.

Under certain conditions not infrequently encountered in practice there will appear, however, as shown above, variations in voltage value of the spark-gap factor of this formula and consequent discrepancies between the actual roentgen-ray output and the estimated dose. But in its routine use for fractional unit dosage as administered by most dermatologists, dosage discrepancies due to the causes just mentioned will, in general, wholly escape recognition, partly because the discrepancies are often small, partly because in fractional unit dosage we have no practical means of ascertaining the amount of roentgen ray administered at any one sitting, and partly because doses much different from those supposedly administered are often followed by highly satisfactory results, apparent proof that the intended fractional amount was received.

With the administration of full unit doses, however, dosage discrepancy will be revealed promptly by the absence or excess of characteristic skin reactions, epilation or erythema; and it was just this experience that drew my attention to this subject, led me to examine the sparkover dosage relations in my own technic and then sparkover dosage relations in general.

$$\frac{6 \times 2 \times 3}{8^2} = \frac{36}{64} = 1 \text{ unit}$$

6 inch gap at 5,280 feet elevation = 80 kilovolts

80 kilovolts at sea level = 4¼ inch point-gap

$$\frac{4\frac{1}{4} \times 2 \times 3}{8^2} = \frac{25.5}{64}, \text{ or about 70 per cent of 1 unit}$$

Dosage, however, does not vary directly as the voltage, but directly as the square of the voltage.

$$36 : 18 + :: 1 : X$$
$$36X = 18$$
$$X = .5 \text{ or } \tfrac{1}{2} \text{ unit}$$

Discrepancies in Dosage Resulting from Use of Standard Formula with Six Inch Sharp Point-Gap at Different Altitudes, Assuming Other Conditions Remain the Same

Altitude			
Sea level	6 inch gap =	100 kilovolts =	1.0 unit
1,000 feet elevation	6 inch gap =	96.3 kilovolts =	0.91 unit
2,500 feet elevation	6 inch gap =	91.3 kilovolts =	0.75 unit
5,000 feet elevation	6 inch gap =	84.1 kilovolts =	0.56 unit
7,500 feet elevation	6 inch gap =	78.7 kilovolts =	0.45 unit

MacKee and Andrews [3] have shown by means of the iontoquantimeter

3. MacKee, G. M., and Andrews, G. C.: Coolidge Tube Quantitative Variations, Am. J. Roentgenol. **11**:525 (June) 1924.

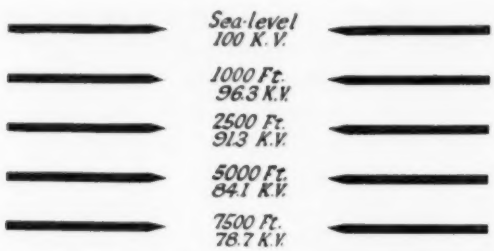

Fig. 1.—Variation in voltage of sparkover across 6 inch sharp point-gap at different altitudes, assuming other conditions remain the same.

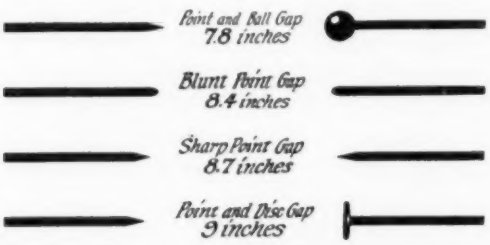

Fig. 2.—Variation in sparkover of 100 kilovolts of current across different types of gap, other conditions remaining the same.

Fig. 3.—Variation in sparkover of 100 kilovolts of current across sharp point-gap at different altitudes, assuming other conditions remain the same.

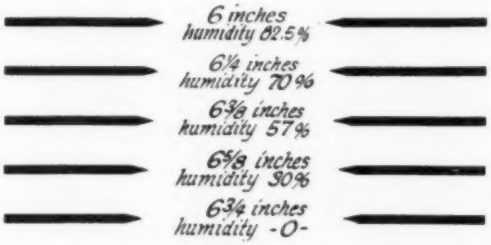

Fig. 4.—Variation in sparkover of 100 kilovolts of current across sharp point-gap at different degrees of humidity, other conditions remaining the same.

that various Coolidge tubes may reveal a difference in roentgen-ray output reaching percentages as high as 57.8 for unfiltered radiation, yet clinical results with these same tubes operated under identical conditions were little different from those ordinarily observed in practice.

It is, of course, conceivable that the mathematically derived dosage discrepancies shown above might similarly be unrevealed in clinical results—a matter for future consideration. Yet, voltage being the prime factor in roentgen-ray production, it seems certain that voltage variations to the extent just considered will result in variations in roentgen-ray output that will be manifested not only mathematically but also therapeutically.

It is then the sole purpose of this brief paper to show that in roentgen-ray therapy, carried out as it now is, under widely varying conditions of altitude, climate and mechanical perfection, the use of the point-gap sparkover to estimate voltage is unreliable, misleading and conducive to great confusion in dosage and technic.

It may be replied that the indirect method and spark-gap estimation of voltage have been employed for many years with great satisfaction, but it should be noted that this method was developed, was introduced, and has been most widely used by workers at or near sea level where sparkover variations are least evident and least troublesome: Up to 1,000 feet elevation the effect on dosage of sparkover variations may not be important, yet, it is not wholly negligible; but in many other localities and under many other conditions dosage discrepancies appear that are inconsistent with satisfactory therapeutic results.

In a paper on an allied subject, Andrews [4] has recently discussed voltage variations arising from lack of uniformity in spark-gap design and from various other structural inconsistencies in apparatus, conditions that are well known and wholly remediable, but the joint influence of air density, humidity and corona on the length of point-gap sparkover not only results in greater and irregular variations in voltage but also is neither calculable nor remediable.

The indirect method of estimating superficial unfiltered roentgen-ray dosage is, as previously stated, a most valuable and practical asset to dermatologic therapy. The standard formula by means of which this method is generally applied is satisfactory within certain limits, but for reasons outlined above this formula is not adapted to universal use. However, it could be made actually standard by a simple change in a single factor, that is, the determination of the voltage necessary to produce the skin unit and the use of that voltage under all conditions whether it induced a 6 inch (15 cm.) or a 10 inch (25 cm.) sparkover.

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4. Andrews, G. C.: Units in Superficial Roentgen Therapy, Am. J. Roentgenol. **14**:336 (Oct.) 1925.

At present, voltage determinations are best made by use of a sphere-gap, either as a permanent part of the equipment or to calibrate each machine for such various combinations of voltage and milliamperage as may be desired.[5]

The use of the sphere-gap for this purpose is, as is well known, based on its freedom from practically all the defects of the point-gap, corrections being required only for alterations in air density which are thoroughly understood.

A practical phase of this subject, however, is the capacity of roentgen-ray machines to deliver the output at which they are rated by the manufacturers; a 2 kilowatt transformer is usually stated to deliver the equivalent of a 7 inch point-gap sparkover; at sea level it will, of course, do so, but at higher altitudes this 7 inch (18 cm.) spark represents an increasingly lower voltage and makes it impossible to obtain under such conditions the output of roentgen ray required by modern technic.

Purchasers of equipment should be fully aware of the conditions under which they operate, and adapt their apparatus to meet these conditions.

DISCUSSION

Dr. Lloyd W. Ketron, Baltimore: It appears that the various extraneous factors tend to cause a smaller dose to be given than the formula indicates. I should like to know whether any factors may arise which would cause a larger dosage than is indicated by the formula.

Dr. George M. McKee, New York: Dr. Markley has called attention to an important source of error in roentgen-ray therapeutic technic, an error that is embarrassing to physicists as well as to roentgenologists. I agree with practically every point mentioned in Dr. Markley's scientific and valuable paper.

The technic in use today, while being sufficiently reliable for practical purposes, is far from precise when the radiation is measured in a well equipped laboratory by a properly trained physicist. Anything that will simplify technic, or will make it more accurate, should be enthusiastically welcomed. There are a number of factors that give rise to error. Dr. Markley has confined himself to one of these; namely, the voltage as estimated by spark-gap lengths. Any variation of voltage during a treatment, or any miscalculation of voltage, may cause an important dosage error. As pointed out by Dr. Markley, we now know that intensity or quantity varies in proportion to the square of the voltage, not in proportion to the voltage, as was formerly believed. This means that if the length of the spark-gap, or voltage, is doubled, the dose instead of being doubled will be quadrupled. Spark-gap estimation of voltage is inaccurate for many reasons. In the first place, there is no standard spark-gap. Some apparatus is equipped with a sharp point-gap, others are equipped with dull points and others have a sphere-gap, and the spheres are both large and small. When dealing with comparatively low voltages, I have found sharp points preferable. Temperature, humidity, ventilation, dust and especially altitude cause errors when estimating voltage by means of the spark-gap.

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5. Ullman, J. H.: Am. J. Roentgenol. **8**:195 (April) 1921.

In the past there has been considerable controversy regarding the relative values of the spark-gap and the voltmeter. The voltmeter was always used in laboratories, but until recently it was not considered practicable for therapeutic work. Now, however, it is possible to mount voltmeters and milliampere meters on the switchboard together with the other necessary controls, and most modern apparatus is so equipped.

However, the voltmeter does not record voltage under all conditions accurately. It must be calibrated by means of the spark-gap when operating on definite milliamperage. If the milliamperage is changed the voltmeter must be again calibrated. Of course, it is possible to have any number of calibrations so that the operator may employ different amounts of current. As this is confusing and likely to lead to error, it seems preferable for the operator to standardize his technic and employ one or perhaps two calibrations.

Because the voltmeter must be calibrated by the spark-gap it is possible to make many of the errors that are associated with spark-gap estimation, but after the voltmeter has been correctly calibrated it becomes a fixed factor that will not change with altitude, humidity, etc., although, like any other mechanical or electrical device, it may get out of order. For this reason it may be advisable to have two voltmeters as well as two milliampere-meters. It might be well also to state the conditions under which the calibration was made — exact length of spark-gap, type of spark-gap, effluve or jump-over spark, milliamperage, type of apparatus, humidity, temperature, altitude, etc.

The voltmeter should now be considered an essential instrument of precision, but it is advisable not to discard the spark-gap—at least not yet. What appears to be required is standardization of apparatus, spark-gap, instruments of precision, calibration of voltmeters, methods of estimation and, in short, the entire technic. This will probably be done by a suitable committee of physicists and roentgenologists.

Arithmetical computation is not suitable for universal use. It is subject to errors of physics, mathematics and judgment. It should be used only by experts, and even then only with caution, at least until it and all other technical factors have been definitely standardized.

The beginner, basing his technic on the skin unit formula of Remer, should test such technic on his own skin, or on mice as advocated by Hazen, before attempting therapeutic work. I agree with Dr. Markley that this formula should be restandardized, so that it will include both spark-gap and voltmeter readings.

Dr. Henry H. Hazen, Washington, D. C.: Much has been said at this meeting about the various errors that are inherent in the arithmetical method of MacKee. On the other hand, I will report a number of results that tend to show the safety of the method. I have been interested for the last two years in estimating roentgen-ray dosage by means of the biologic effect. The method we have used has been the epilating dose on mice. The work has been done entirely with one tube, under relatively constant humidity and at the same elevation. It has been a notable fact that if a 5¾ unit dose is given epilation is not complete in three weeks. If 6 units are given the epilation is complete in three weeks. This method holds not only for the standard spark-gap of 7¾ inches (19.7 cm.) but also for those of any size from 4 to 10 inches (10 to 25 cm.). We have worked this out at various distances also, and apparently the arithmetical method is constant within a small fraction of error, using one machine and with a constant humidity. It seems to me that a method of this type is simple, and I can see no reason why a biologic epilation method on mice will not

sooner or later be a method of standardizing machines, just the same as it should be in standardizing radium.

Dr. A. J. Markley, Denver: In reply to Dr. Ketron it will be noted that these formulas were worked out at sea level. The only change, therefore, that would result in increase of dosage would be a great increase in humidity with consequent shortening of the spark-gap, which, if then lengthened to comply with the standard, would increase the dose.

Dr. MacKee stated that the sphere-gap is subject to the same disturbing influences as the point-gap. This, I am certain, is a mistake. The sphere-gap sparkover is influenced only by alterations in air density, the resulting changes occurring in a definite ratio and being readily compensated.

Dr. MacKee assumed that I was advocating the determination of voltage by means of the kilovolt meter — no, it has the same inaccuracies as the point-gap. The best means of voltage measurement at present is the sphere-gap which should be attached at the tube terminals.

Dr. Hazen has developed an entirely individual technic and formula using a 7½ inch (19 cm.) point-gap sparkover, but, as shown above, its use will produce a totally different amount of roentgen ray if used under conditions different from those in Washington.

A plea has been made that manufacturers standardize the construction of the point-gap, but a general recognition of its total unreliability in the estimation of voltage is leading manufacturers to abandon the use of the point-gap altogether.

A definite amount of voltage must be established, which being passed through a Coolidge tube, will produce a definite quantity of roentgen ray at sea level or elsewhere, although it may induce a widely varying point-gap sparkover.

FIXED ARSPHENAMINE DERMATITIS *

HENRY H. HAZEN, M.D.
WASHINGTON, D. C.

In the English and American literature there is a striking paucity of references to the so-called "fixed arsphenamine dermatitis," cases having been reported only by Moore and Keidel [1] and Stokes and Cathcart.[2]

The first description of this condition was given by Naegeli [3] in 1917. Following this article there came similar reports from Fuchs,[4] Engweer and Josephson,[5] Thibierge and Mercier,[6] Schönfeld,[7] Leibkind,[8] Kraus [9] and Levin.[10]

The patient who is the subject of the following case report was seen by my associate, Dr. F. J. Eichenlaub, in a clinic in 1920 and was brought to the office by him for observation.

REPORT OF CASE

A white woman, aged 34, sought relief, in 1920, for a circinate nodular syphiloderm situated on the outer aspect of the right forearm about midway between the wrist and the elbow. The Wassermann reaction was strongly positive. Following the fourth injection of arsphenamine the fading syphilitic lesion suddenly became converted into a typical weeping dermatitis. Three more injections were given and each time the lesion became intensified, only to fade in the intervals between injections. Four months later arsphenamine was again tried, but because of the severity of the dermatitis was discontinued after only two injections were given. Two years later the patient was again seen at the Georgetown University Hospital Clinic, and once again the same experience was encountered. Curiously enough, only a few weeks ago this patient again reported with an exactly identical dermatitis, although she said there had been no administration of arsenic in any form.

The lesions always developed on the site of the previous syphilid, and on no other portion of the body. There were no evidences of anaphylaxis or hyper-

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* Read at the Golden Anniversary Meeting of the American Dermatological Association, Philadelphia, May, 1926.

1. Moore, J. E., and Keidel, A.: Dermatitis and Allied Reactions Following Arsenical Treatment of Syphilis, Arch. Int. Med. **27**:716 (June) 1921.
2. Stokes, J. H., and Cathcart, E. P.: Postarsphenamine Dermatitis, Arch. Derm. & Syph. **7**:14 (Jan.) 1923.
3. Naegeli: Cor.-Bl. f. schweiz. Aerzte. **47**:1276, 1917.
4. Fuchs: Deutsche med. Wchnschr. **45**:1276, 1919.
5. Engweer and Josephson: München. med. Wchnschr., Feb. 28, 1919, p. 243.
6. Thibierge and Mercier: Bull. Soc. franç. de dermat. et syph. **93**, 1919.
7. Schönfeld: Deutsche med. Wchnschr. **46**:11, 1920.
8. Leibkind: Dermat. Ztschr. **31**:91, 1920.
9. Kraus: Deutsche med. Wchnschr. **46**:913, 1920.
10. Levin: Dermat. Wchnschr. **72**:278, 1921.

susceptibility except the fact that there was a recurrence of increasing severity following each administration of arsphenamine. Unfortunately, an eosinophilia was not demonstrated, nor was there any attempt to demonstrate a local hypersensitiveness to arsphenamine.

A study of the other reported cases reveals the fact that no one drug was responsible and that reactions followed the third and fourth injections rather than the first. In Naegeli's patient the injection of epinephrine hydrochloride prevented the development of the lesion. Jadassohn's [11] patient was made much better by the administration of epinephrine hydrochloride. Stokes' patient later developed an exfoliative dermatitis.

Injection of the conjunctiva was noted in some cases. In several instances there were multiple lesions rather than solitary spots. There was no predilection for any one area.

The genesis of this condition is interesting. There seem to be two tenable theories, first a local hypersensitiveness, second a Herxheimer reaction. A purely local hypersensitiveness is in strict harmony with the conception of anaphylactic phenomena in general. Lucas and Gay,[12] among others, have apparently settled this point. The fact that none of the reactions has followed the first injection and that epinephrine hydrochloride has been of aid would seem to point to the hypersensitive theory as being the correct one. The similarity of this eruption to the fixed phenolphthalein eruption naturally favors the same view.

DISCUSSION

Dr. Herman Goldenberg, New York: I fear that Dr. Hazen has overlooked the fact that four cases of fixed arsphenamine eruption have been presented from my service. The first, in 1918, was probably the first case presented in America. At that time it was remarked by some of the physicians present at the demonstration that the case closely resembled a phenolphthalein eruption and that this drug was the cause of the eruption. We subsequently administered phenolphthalein in this case but could not reproduce the eruption, which we were able to do by again administering arsphenamine. Since then I have had three other cases presented, two by Dr. Chargin and one by Dr. Levin. In one case the eruption followed the twentieth injection, in another the thirty-first and in the third the seventeenth.

Dr. Fred Wise, New York: I am almost convinced that the recurrence of an eruption such as Dr. Hazen described can be explained by the fact that the patient took either phenolphthalein or antipyrine. Either of these drugs will produce an eruption in situ in the area which was formerly the site of an arsphenamine eruption.

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11. Jadassohn, quoted by Fuchs (footnote 4).
12. Lucas and Gay: J. M. Research, 1909, p. 251.

URTICARIA PIGMENTOSA *

LOUIS B. MOUNT, M.D.
ALBANY, N. Y.

In the *British Medical Journal* of Sept. 18, 1869, the following case report appeared:

Emilie P., aged two years, living at Blackheath Hill, was admitted at the Hospital for Skin Diseases on July 27, 1860. She was the subject of chronic urticaria, and the eruption was peculiar in leaving stains of a light brown colour, their tint being very like that of chloasma for which the disease might easily have been mistaken at first glance. She had light hair and a very fair complexion. The mother says that the child "has never suffered from any illness since her birth"; and she is in excellent health. There was no history of urticaria in other members of the family. The present eruption began when she was three months old and she has never been free from it since. The mother's account is, that the spots began as "white lumps like the sting of a nettle;" these itch severely, and, on subsiding leave the curious brownish stains above noticed. The rash, on admission, thickly covered the neck and trunk, the extremities being more sparsely affected. There were no spots on the face, but a few brown stains of former wheals at the margin of the scalp on the forehead. There were no red wheals, but some slightly raised patches of light brown colour with slight congestion, and some stains of former wheals. The true nature of the raised patches was proved by their entire centres turning nearly white when the skin was stretched. The wheals are of uniform size, and about as large as three penny pieces. It was noticed that a scratch with the finger nail produced, in a few minutes, an ordinary urticarial wheal, with white centre and red edges; and, at a subsequent occasion, a number of recent patches of elevated and erythematous skin were observed; these had not yet become brown. When the child first came there was little or no evidence of scratching; but, several weeks afterwards, it is noted that, "in addition to the urticaria, there are scratched papules, like those of prurigo; no lice can, however, be found." At her last attendance five weeks from admission, she was in much the same condition as at first, excepting the pruriginous spots.

We do not find any mention of a similar condition in our standard works.

Mr. Nettleship called this "chronic urticaria, leaving brown stains; nearly two years' duration."

It is from this most lucid description that the recognition of urticaria pigmentosa, the name given to the condition by Sangster in 1877, has been made possible.

Thin [1] in 1875, and Fox [2] and C. and F. E. Hoggan,[3] in 1883, gave the first results of histologic examinations in infantile cases. The Hoggan brothers irritated a lesion and then excised it. They found a

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* Read at the Golden Anniversary Meetnig of the American Dermatological Association, Philadelphia, May, 1926.

1. Thin: Tr. Clin. Soc. London, 1875.
2. Fox, C.: Med. Chir. Tr., **66,** 1883.
3. Hoggan, C., and Hoggan, F. E.: Monatsh. f. prakt. Dermat. **2:**44, 1883.

massive collection of cells in the papillary portion of the cutis. There were so many that they compressed each other into cuboid shapes and were arranged in parallel columns. The cells were large and, as the examiners thought, edematous. They believed them to be white corpuscles due to the mechanical irritation of the lesion. Unna [4] later identified these cells as Ehrlich mast cells and also showed that the edema was not intracellular but rather extracellular. To the papule of urticaria pigmentosa he gave the name of mast cell tumor.

Raymond,[5] in his masterful monograph on the subject, made a critical survey of all the available material, and separated the disease into three clinical periods: 1. Numerous wheals appear in various crops, remain a while, retrogress, and leave either brown spots or papules. The duration of this period is from a few days to a year. 2. Recrudescence of the urticaria occurs in these spots and papules, with no real increase in the number of wheals. This goes on for from two to five years. 3. The crops become fewer; the disease retrogresses and ends with restitutio ad integrum in from four to six years more. Raymond also stated that he was convinced of the connective tissue origin of mast cells, and described degenerative forms.

After Raymond's article, there appeared numerous case reports and many excellent papers, notably those of Baumer,[6] Blumer,[7] Rona,[8] Doutrelepont,[9] Nobl,[10] Reiss,[11] Fabry,[12] Little,[13] Knowles,[14] Hartzell,[15] Wise,[16] Finnerud [17] and Hannay.[18]

CAUSE OF URTICARIA PIGMENTOSA

The exact cause of urticaria pigmentosa is shrouded with mystery. There have been but a few instances of any familial tendency. Wallace-

4. Unna: Dermat. Studien, 1887.
5. Raymond: L'Urticaire pigmentee, Paris, O'Doin, 1888.
6. Baumer: Arch. f. Dermat. u. Syph. **34**:323, 1896.
7. Blumer: Monatsh. f. prakt. Dermat. **34**:213, 1902.
8. Rona: Arch. f. Dermat. u. Syph. **40**:13, 1897.
9. Doutrelepont: Arch. f. Dermat. u. Syph. **22**:311, 1890; Monatsch. f. prakt. Dermat. **9**:372, 1889.
10. Nobl: Arch. f. Dermat. u. Syph. **75**:163, 1905.
11. Reiss: Monatsh. f. prakt. Dermat. **37**:93-111, 1903.
12. Fabry: Arch. f. Dermat. u. Syph. **113**:269, 1912.
13. Little: Brit. J. Dermat. **17**:355, 393 and 427, 1905; **18**:16, 1906.
14. Knowles: J. Cutan. Dis. **33**:171, 1915.
15. Hartzell: J. Cutan. Dis. **35**:756, 1917.
16. Wise: Interstate M. J. **25**:52, 1918.
17. Finnerud, C. W.: Urticaria Pigmentosa (Nodular Type) with Summary of Literature, Arch. Dermat. & Syph. **8**:344 (Sept.) 1923.
18. Hannay: Brit. J. Dermat. **37**:1, 1925.

Beatty [19] stated that the affection had occurred in two brothers; Darier,[20] in three sisters and Little,[13] in a mother and cousin of one of his patients. It has been attributed to pregnancy late in life, to vaccination, to a sulphur bath, to fright, to chickenpox, to measles, to insect bites, to maternal shock during pregnancy, and to a host of other incidents with or without any rime or reason.

Neisser [21] states:

> I have not the slightest doubt but that in urticaria pigmentosa we have to do with "mastzellentumoren;" but how explain the connection between the urticaria and the tumor like formation? I believe that the two conditions, urticaria and tumor like formation, are two different things each for itself, and a causal communication between them is not established. The tumors are of an inherited form and disposition, like the embryo deposits in tumors. The urticaria is only the chance cause of these apparitions.

Little [13] thinks the condition is a congenital abnormality, partly because of its onset in so many cases early in life, and partly because he had demonstrated, in two of his cases, an infiltration of mast cells in clinically normal appearing areas of skin. Gilchrist [22] also reported a similar observation.

Welch [23] suggests that there is a toxemic state of the blood and that when the skin is stimulated, toxins are set free in the tissues.

Brongersma [24] declares that the mast cells are already present in the tissues long before a wheal has appeared. The mast cell granules, lying free in the lymph spaces degenerate, enter the blood stream, act as a toxin exciting changes in the superficial circulation and producing urticaria. The changes incident to wheal formation, edema, etc., sweep the most cells together.

Knowles [14] says that a congenital abnormality of the skin has apparently been proved in cases of urticaria pigmentosa by the finding of mast cells in the clinically normal skin of those with this condition. A congenitally abnormal skin is therefore much more easily acted on than is one that is normal. Some toxin, apparently, which possibly might not prove to be deleterious to a normal skin is evidently causal in this condition. The nature of the toxin or the way in which it acts is as yet unknown.

Paramore,[25] from experimental work in this disease, arrives at the conclusion that there is a definite modification in the red blood corpuscles

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19. Wallace-Beatty: Dublin J. M. Sc. **121**:422, 1906.
20. Darier: Ann. de dermat. et syph. **2**:339, 1905.
21. Neisser, quoted by Little (footnote 13).
22. Gilchrist: Bull. Johns Hopkins Hosp. **7**:140, 1896.
23. Welch, quoted by Gilchrist (footnote 22).
24. Brongersma: Brit. J. Dermat. **11**:179, 1889.
25. Paramore: Brit. J. Dermat. **18**:24, 1906.

tending to increase their resistance to destructive agents. The salt content of the blood is increased, the content in lime salts is at least not diminished. That urticaria may in some cases be due to a modification in the salt content of the blood is possible, but in view of the fact that all the characteristics found in the blood in urticaria pigmentosa have also been found in cases of hemophilia, purpura, etc., he seems to feel that it is plausible to consider the disease as more akin to the so-called blood disorders, or perhaps occupying an intermediate position between the purpuric condition and the urticarias.

Heimann [26] is inclined to think, from the fact that the majority of cases of urticaria pigmentosa occur very early in infancy, that a great many are undoubtedly congenital; also that they recur temporarily in the sense of nevi. He feels that the acceptance or nonacceptance of the classification of any congenital anomaly of the skin as a nevus is problematic. The adult type he regards as a latent form of nevus.

THE DISEASE PICTURE

At first Unna [27] considered mast cells to be emigrated wandering cells, but later he decided that they were connective tissue cells which had taken up mast cell granules and did not multiply by cell division, as Quinquaud and Nicolle [28] declared, but increased in number by the addition of transformed spindle cells. Both Little [13] and Ranvier [29] believe that these cells are derived from the blood because of the fact that they are found surrounding the blood vessels. Neither Gilchrist [22] nor Brongersma [24] subscribe to their blood derivation, the former because of the finding of numerous mast cells in a wheal four minutes after its formation, the latter because no investigator had ever detected them in the vessels.

The mast cell papule, according to present opinion, does not appear on the scene as a completed picture. Every new urtication causes an increase in mast cell material which remains as a solid remnant of the acute periods. The constant repetition of the process ultimately converts the papillary portion of the cutis into a mast cell tumor.

The microscopic findings, in cases beginning in the early years of life, are fairly constant. These are, in the main, an increased amount of pigment in the epidermis and a varying grade of infiltration of the corium with mast cells. The mast cell infiltration is usually in the uppermost portion of the corium, that is, in the papillary and subpapillary zones, and is so dense that the cells are squeezed and pressed into odd

26. Heimann: J. Cutan. Dis. **35**:763, 1917, discussion of Hartzell's article (footnote 15).

27. Unna: Histopathology of Diseases of the Skin (transl. by Walker), 1896.

28. Quinquaud and Nicolle: Ann. de dermat. et syph. **2**:393, 1891.

29. Ranvier, quoted by Little (footnote 13).

shapes. Their arrangement is in distinct bands and columns, with collagen bundles in between. The basal cell layer is flattened due to underlying pressure. The lower layers of the rete is the seat of the increased pigmentation, and some pigment may be found in the upper part of the corium.

Variations from this picture are known. Quinquaud [30] observed in children some cases of pigmentary disease of three years' duration. The search for mast cells was fruitless. Michelson's [31] patient, an infant of 4 months, showed an infiltration in which there were but few mast cells. Rona saw heaps and rows of cells, partly mononuclear leukocytes, partly plasma cells, with dilatation of the lymph and blood vessels. Doutrelepont [32] described microscopic observations from three excisions of lesions of varying ages, which were not those of a mast cell tumor. Jadassohn [33] also found, in one of his juvenile cases relatively few and disseminated mast cells. Thus with but comparatively few recorded exceptions, the most frequent observation is the mast cell tumor of Unna.

Liveing, at the International Congress of 1881, reported the first recorded case of the adult type, the patient being a 14 year old girl with a macular pigmented eruption of one and a half years' duration.

An analysis of eighty-seven recorded instances in which the eruption had its inception at or later than the age of 13, shows that 37 per cent began between the ages of 13 and 20, 30 per cent between 20 to 30, 19 per cent between 30 to 40, 10 per cent between 40 to 50 and 4 per cent after 50. Fifty-seven per cent had a macular eruption, 8 per cent a nodular eruption and 35 per cent a maculonodular or mixed eruption. From this it can readily be seen that the majority, 67 per cent, of the so-called adult cases begin between the ages of 13 and 30, and that the predominating type of eruption is the macular.

In their symptomatology, they differ little from the infantile or juvenile cases. Adamson [34] and others have said that in no case have been seen the large macules or nodules characteristic of the urticaria pigmentosa in children. Just how far this statement is true is difficult to ascertain, because some of the recorded instances are almost similar, according to their descriptions, to the condition in juveniles. Itching varies considerably, exactly as it does in the infantile type. The

30. Quinquaud: Ann. de dermat. et syph. **4**:859, 1893.

31. Michelson, H. E.: Urticaria Pigmentosa, Arch. Dermat. & Syph. **6**:644 (Nov.) 1922.

32. Doutrelepont: Arch. f. Dermat. u. Syph. **22**:311, 1890.

33. Jadassohn: Verhandl. d. deutsch. dermat. Gesellsch. **4**:380, 1894.

34. Adamson: Brit. J. Dermat. **35**:219-222 (June) 1923.

coalescence of macules into quite large plaques has been noted, also vesicles on the tops of small nodules, the latter by Pusey.[35]

When one considers the pathologic condition in the reported adult type cases, he is at once impressed with the great variability in the microscopic observations. These vary all the way from no mast cells to mast cell tumor. The statement "no mast cells found" is not so uncommon; also "some mast cells found," meaning by this term comparatively few. Should this criterion, unaccompanied by other information, be accepted as final?

Quinquaud and Nicolle [28] state: "It is not therefore the presence of mast cells which must be regarded as pathognomonic, but their exclusive presence in considerable numbers." Heimann [26] said, "The isolation of a mast cell here and there was not enough to make a diagnosis of urticaria pigmentosa."

The degeneration of mast cells is well known and nicely portrayed by Raymond [5] in the following words:

> To the two microscopic stages of the lesions, the one prominent and deep-colored, the other flattened and paler, correspond, I believe two different microscopic conditions. In the one the protoplasmic cells are numerous, living, and in process of formation; in the other they are no longer produced, but die and disintegrate, and then are absorbed.

Hartzell [15] and Wise [16] also comment on mast cell degeneration. But can mast cell degeneration explain all the known facts? Can it explain facts such as these: very few mast cells even though for many years urticarial crops occurred and were occurring at the time of excision (Blumer's [7] case); mast cell tumor years after cessation of the crops (Blumer's [7] case); disseminated mast cells many years after cessation of wheals as in Jadassohn's [33] case, in which for fourteen years no spontaneous eruptions or subjective symptoms occurred?

What seems to be of considerable importance is the presence or absence of marked evidence of inflammation. In the face of its presence, how can a diagnosis of urticaria pigmentosa be arrived at? Such cases ought to be considered as urticaria with pigmentation even though a few mast cells might be found.

The following case of mine is illustrative of the foregoing.

> A woman, aged 38, whose family and past personal history were not important, sought aid because of an itching eruption of a year's duration. She presented, on various parts of the body, the face and neck excepted, urticarial wheals, also brownish macules, finger-nail in size. Some of the wheals appeared bluish red, and the color could not be dispelled. Dermographism was present. Histologic examination of the wheal determined the cause of the color to be hemorrhage. A macule showed an increase in the pigment of the lower rete, edema and a

35. Pusey: J. Cutan. Dis. **35**:756, 1917, discussion of Hartzell's article (footnote 15).

marked perivascular and periglandular infiltration with polymorphonuclear neutrophils. The clinical diagnosis was urticaria pigmentosa, but the microscope changed that into urticaria with pigmentation.

CONCLUSIONS

Infantile cases, as a rule, present no difficulties in arriving at a satisfactory conclusion.

Adult cases, on the contrary, are problems. I believe that many recorded cases are not examples of the adult type of urticaria pigmentosa but rather of urticaria with pigmentation.

DISCUSSION

Dr. Fred Wise, New York: I should like to report an interesting case in which two biopsy sections were removed at intervals of one year. The first showed no mast cells, but the second showed a well developed infiltration.

Dr. Walter J. Highman, New York: This subject was interestingly dealt with in the last paper Dr. Hartzell read before this body, in 1917. Many errors have crept into our attitude toward urticaria pigmentosa. We are talking of it as a definite clinical entity. The question of whether the mast cells are present or absent is a matter of when the lesion is examined, and whether a proper selection was made for microscopic investigation. In the early stages there are few mast cells. At the height there are many of them, and then they gradually disappear as the lesion involutes. That being the case, so far as urticaria pigmentosa is concerned we have a definite point of departure. The other type which develops later in life may be classified in two groups, either urticaria pigmentosa in the strict sense or urticaria with pigmentation. I think Brocq pointed this out. When mast cells are found in profusion, I think it is a matter of chemotaxis; certainly these mast cells are not found in the group of erythematous multiform lesions. The presence of mast cells has something to do with the matter of local nutrition, so I do not think we can say that the mast cell is necessarily a diagnostic feature in urticaria pigmentosa unless it is present in great numbers, when it is pathognomonic. I think Dr. Mount was very wise in emphasizing this point.

Dr. Louis B. Mount, Albany: The observations are contradictory. In the adult cases scattered mast cells have been found years after all of the urticarial element has disappeared. They have also been found as a solid mast cell tumor years after the other symptoms have disappeared, so that while the question of degeneration of mast cells may enter into the problem, I do not see, as I stated in my paper, how that can answer all the questions which arise in going over and analyzing a group of these adult cases.

Editorial

ULCUS VULVAE ACUTUM AND HERPES VULVAE

Dr. Clark W. Finnerud has brought ulcus vulvae acutum to attention in America. There is a considerable resemblance between this newly recognized condition and herpes vulvae. The symptoms in many respects are the same as those of herpes. *Bacillus crassus* is a saprophyte which becomes pathogenic in these cases. Its rôle in this condition is not absolutely established. Can it be that ulcus vulvae acutum is an exaggerated form of herpes of the genitals? It is important that these facts be made clear.

With these questions in mind, the matter was referred to Dr. Finnerud in the following memorandum:

The resemblances to herpes of the genitals are as follows: (1) the onset is sudden, (2) the lesions are very painful, and (3) their recovery is spontaneous and rapid. In addition the ulcers are small, from 2 to 7 mm. in diameter, oval and round. They are not grouped. They are more numerous than is usual in herpes. Their greater number (though herpes may be abundant) and their lack of grouping (if that is a fact) are not like herpes. The other characteristics are certainly difficult to distinguish from herpes.

Dr. Finnerud's reply is as follows:

You state, "The resemblances to herpes of the genitals are as follows: (1) the onset is sudden, (2) the lesions are painful, and (3) their recovery is spontaneous and rapid." These points are beyond dispute and constitute the chief and, I believe, practically the only resemblances to herpes. You then say: "In addition, the ulcers are small, from 2 to 7 mm. in diameter, oval and round." This is true only of one type of ulcus vulvae acutum, that characterized by small lesions. Usually when the lesions are small, they are more numerous and more superficial. This group makes up more than half of the cases and it is this group that clinically somewhat resembles herpes.

In the "gangrenous type" the lesions are few and particularly large and deep. Nearly half the cases are of this type.

The lesions of ulcus vulvae acutum are commonly ushered in with chill and fever and the lesions are deeper and usually larger than in herpes, and, because of their depth, they often leave scars. As you say, they are without much tendency to grouping. The ulcers are often undermined, with irregular, even serrated margins and are sometimes fissure-like, though more commonly the lesions are round or oval. The lesions do not begin as vesicles and vesicopapules. The subsequent lesions are not merely erosions but true ulcers, and these do not tend to group in the form of circle segments, as is often the case in herpes.

The ulcers are commonly much more destructive than in my case, but you will notice that one of the three is rather deep.

I shall report the bacteriologic observations in the first case of herpes of the female genitals that I see.

Dr. Finnerud by this statement, it would seem, establishes the case for ulcus vulvae acutum. It would not be without interest, however, to study the bacteriology of herpes of the genitals.

W. A. P.

News and Comment

CALIFORNIA

Another Fatal "Face Peel."—The coroner's jury held that the death of Mrs. Louise Wulbers, San Francisco, September 9, was the result of shock, excitement and absorption of carbolic acid while undergoing a "knifeless facial operation" at the hands of "Dr." Zailick Saltzman. This office has no record of a doctor of medicine by that name. The California State Board of Medical Examiners notes that Mrs. Wulbers died suddenly following the application of phenol solution to her face; also that the records of the board show that several other deaths have been reported as due to the absorption of phenol applied by so-called beauty specialists during the operation known as "face peel."

ILLINOIS

Promotion at Rush Medical College.—The board of trustees of the University of Chicago recently announced the following promotion in the faculty of Rush Medical College:

Dr. Michael H. Ebert, clinical associate, department of dermatology.

INDIANA

Personal.—Dr. Chester N. Frazier, an associate in dermatology at Peking Union Medical College, Peking, China, who has been granted a year's leave of absence, is visiting his parents at Bluffton.

Society News.—Dr. Frank W. Cregor, Indianapolis, addressed the Jasper-Newton County Medical Society, August 2, at Rensselaer, on "Common Skin Diseases."

MASSACHUSETTS

First Chair in Dermatology at Harvard University.—The following is a facsimile of a letter making an appointment to the first chair in Dermatology at Harvard University.

Harvard College.
Cambridge, Nov. 2d 1871.

Dear Sir

I have the honor to inform you that on the 19th of October 1871 you were appointed by the President and Fellows, Professor of Dermatology in the Medical School for the term of five years from September 1st 1871. and that this appointment was duly confirmed by the Board of Overseers at a meeting held by them this day.

Yours truly
Charles W. Eliot
President

James C. White, M. D.
No. 10 Park Square

OHIO

Promotions and Appointments at Western Reserve University.—The following promotions and appointments to the faculty of Western Reserve University Medical School have been announced in the *Bulletin* of the Academy of Medicine of Cleveland:

PROMOTIONS

Dr. John A. S. Gammel, instructor in dermatology and syphilology for two years.

Dr. John Rauschkolb, instructor in dermatology and syphilology for two years.

APPOINTMENTS

Dr. Clyde L. Commer, demonstrator in dermatology and syphilology for one year.

SMALLPOX

KENTUCKY

Smallpox Epidemic.—An outbreak of fifty-five cases of smallpox with two deaths was reported from the Horse Branch section of Ohio County, September 18; the outbreak is said to have started three weeks previously. In an effort to prevent the further spread of smallpox, attendance at all public meetings in the affected area has been prohibited, it is reported.

NEW YORK

Smallpox Record.—As a result of systematic vaccination, there has not been a death from smallpox in Syracuse for thirty-seven years, according to the city health department. There have been, however, since Jan. 1, 1926, nineteen cases of infantile paralysis in Syracuse with five deaths, also an epidemic of measles of 2,257 cases. There was not a case of measles reported in an infant under 3 months of age.

TEXAS

Smallpox in Texas Increasing.—In the first seven months of this year, eighty-six counties in Texas reported smallpox, the number of cases ranging from 234 in Harris County to one in a number of other counties. The total number of cases reported in that period was 1,668, while for a similar period in 1925 there were 1,228 cases and in a similar period in 1924 there were 857 cases. The state board of health has issued a bulletin stating among other things that the automobile in cross-country travel brings the smallpox menace to every town; health officials are urged to use every means possible to secure vaccination against smallpox.

ITALY

Special Honors Conferred on Dr. A. Bertarelli.—The Consiglio degli instituti ospedalieri of Milan bestowed special honors recently on Dr. Ambrogio Bertarelli, director of the Ospedale Maggiore. Professor Papini, director of the Clinica Dermosifilopatica of the University of Milan, gave the assembled gathering an account of the career of Bertarelli, who has contributed so greatly to the progress of dermatology in Milan. He furnished the impetus that led to the creation of the photoradiotherapeutic department in the Ospedale Maggiore. Two gold medals of merit were conferred on him.

GERMANY

Prof. Dr. Oscar Gans to Lecture for Mayo Foundation.—Prof. Dr. Oscar Gans of the University of Heidelberg, author of "Histologie der Hautkrankheiten," at present is giving a course in histopathology of the skin before the Section on Dermatology, and delivering a series of lectures for the Mayo Foundation at Rochester, Minn. He plans to remain there until approximately Jan. 1, 1927, when he will visit in various cities in this country before his return to Heidelberg.

NATIONAL DERMATOLOGIC SOCIETIES

AMERICAN DERMATOLOGICAL ASSOCIATION

George Miller MacKee, President, 200 W. 59th St., New York.
C. Guy Lane, Secretary, 421 Marlborough St., Boston.

SECTION ON DERMATOLOGY AND SYPHILOLOGY, SCIENTIFIC ASSEMBLY, AMERICAN MEDICAL ASSOCIATION

James Herbert Mitchell, Chairman, 25 E. Washington St., Chicago.
William H. Guy, Secretary, Jenkins Arcade, Pittsburgh.

LOCAL DERMATOLOGIC SOCIETIES

ATLANTA DERMATOLOGICAL SOCIETY

Chas. A. Wilkins, President, Hurt Bldg., Atlanta, Ga.
Jack W. Jones, Secretary, Atlanta National Bank Bldg., Atlanta, Ga.

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Silesian Dermatologic Society.
Association of French-Speaking Dermatologists and Syphilologists.

Abstracts from Current Literature

Degree of Mental Deficiency Resulting from Congenital Syphilis. N. A. Dayton, J. A. M. A. **87**:907 (Sept. 18) 1926.

Dayton reports that the intelligence in sixty-one cases of mental deficiency with congenital syphilis was definitely above that in 1,956 cases due to other causes. When congenital syphilis initiates that pathologic process resulting in mental deficiency the process is less severe, and a higher average of intelligence is attained than in cases with other causation.

From the statistical point of view, congenital syphilis does not produce large numbers of cases of mental deficiency. When we consider the intelligence, congenital syphilis does not produce a low grade of defect. Therefore, from both the quantitative and the qualitative points of view, congenital syphilis is not a serious factor in the production of mental deficiency.

Acrodynia in Adults. C. J. White, J. A. M. A. **87**:1092 (Oct. 2) 1926.

White reports two cases of this disorder; one of them in detail.

The patient was a negro, aged 52, who complained of swelling and burning of the hands and feet, loss of weight, anorexia and other minor symptoms. The hands and feet, and the face in lesser degree, presented exudative areas, and swelling without pitting on pressure. There was also a generalized maculopapular rash. Leprosy, arsenical neuritis, pellagra and beriberi were excluded. Skin photosensitization tests gave negative results. Recovery ensued on a vegetable-fruit diet, symptomatic medication and local applications.

The second case (Carroll S. Wright's) occurred in a white woman, aged 35, who also recovered on simple dietary and symptomatic treatment.

Sulpharsphenamine in the Treatment of Warts. R. L. Sutton, J. A. M. A. **87**:1127 (Oct. 2) 1926.

Sutton found sulpharsphenamine curative in six cases of the juvenile type. Only one intramuscular injection (0.4 Gm.) was required in each case.

Creeping Eruption: Report of First Case from Manitoba. K. J. Austmann, J. A. M. A. **87**:1196 (Oct. 9) 1926.

Austmann reports a typical case in a young adult; the larva was recovered and identified as that of *Gastrophilus intestinalis*.

A review of the reported cases is given, and it is brought out that the cases which present vesicles are probably due to nematodes, while those showing erythematous or papular lesions are probably due to *Gastrophilus larva*.

By means of dermatoscopy the author was able to observe the larva in the skin and to extract it without difficulty.

Leprosy in Korea. R. M. Wilson, J. A. M. A. **87**:1211 (Oct. 9) 1926.

This is a short report of a study of 938 cases. It is estimated that there are from 5,000 to 20,000 lepers in Korea. Anesthesia occurs in 96 per cent of the cases and is the most reliable symptom. Neuritis was found in 26 per cent, "rheumatism" in 58 per cent, fever in 56 per cent, ulceration in 37 per

cent and facial paralysis in 28 per cent of the cases. For treatment, weekly injections of oleum hydnocarpiae, with a 1 per cent solution of camphor added, in doses from 4 to 8 cc., were employed. Hygienic measures were also used. There were 311 deaths in 1,109 cases in the last twelve years. Paroled patients numbered 238, with apparent cure in 30 per cent. Outdoor work, such as gardening, is of real therapeutic value.

MELANOTIC MOLES WITH ESPECIAL REFERENCE TO PROGNOSIS. C. F. LEHMAN, South. M. J. **19**:678 (Sept.) 1926.

Lehman reports eight cases of melanotic moles, two pigmented nevi, a melanosarcoma of the orbit and an extensive "bathing trunk" nevus. The melanotic moles were all studied histologically after removal by cautery. He believes that the amount of contained melanin is no measure of the degree of malignancy. Despite treatment, five of seven patients with malignant melanoma died or became incapacitated; one case was untraced and one patient lived eight years. This type of malignancy should have local removal (the author prefers the cautery) followed by heavy radiation to all involved areas. An unusual case is recorded in which many of the melanotic tumors spontaneously regressed, but finally resulted in the death of the patient.

MICHAEL, Houston, Texas.

EXPERIMENTAL HUMAN HERPES. P. TEISSIER, P. GASTINEL and J. REILLY, J. de physiol. et de path. gén. **24**:271, 301 and 316, 1926.

Herpes vesicle fluid deposited on the scarified skin induced a typical herpes lesion and sometimes a secondary eruption. Tests have been made with the human virus on 400 persons in the last three years. They demonstrated the constant existence of the virus in the herpes vesicle; the susceptibility of man to the virus from an active lesion, even when there was no history of spontaneous eruptions; the inoculability of lesions in series on the same or another subject, and the importance of predisposing physiologic and pathologic factors. As a rule no local or general immunity is conferred, and the virus does not retain its vitality at the point of inoculation; the presence of the herpes virus in the cerebrospinal fluid is exceptional if it ever occurs. Another point brought out by the research is that the presence of the keratitis-inducing virus in the saliva is not closely related to the presence of an existing attack of herpes. The virus applied to the skin works rapidly into the depths, as is evident from the prompt inflammatory reactions discovered in the tissues beneath. Notwithstanding the variety of forms in which clinical herpes develops, the virus from the vesicles always induces identical lesions in animals. The assumption that herpes is the result of a conflict between a morbid cause and the cells of the organism—an inanimate kind of ferment—is sustained by Carrel's recent research on infectious sarcoma in fowls, but the principal argument in its favor is Bordet's refusal to accept the microbic nature of the bacteriophage. The principle responsible for the phenomena does not reproduce itself but is reproduced by the tissues. This conception of an autogenous origin for herpes explains the clinical phenomena better than any other. The virus once generated might lie latent between attacks; this would explain the local recurrences and the affinity of recurring herpes for the nervous system. Discussions of this kind demonstrate the uncertainty prevailing in regard to the ultraviruses; they straddle the still vague borderline between the animate and the inanimate kingdoms.

CHOLESTEREMIA AND GLYCEMIA IN DERMATOSES. LORTAT-JACOB and P. LEGRAIN, Médecine **7**:746 (July) 1926.

The frequent discovery of hypercholesteremia in xanthoma patients and the subsidence of xanthomas in diabetic patients being treated with insulin have suggested the application of insulin in other dermatoses. Testimony is accumulating that the cholesterol content of the blood serum is above the normal level in cases of xanthoma, lipoma and psoriasis, while it is normal or subnormal in eczema and dermatitis. The sugar content of the blood seems to be excessive in pruritus, psoriasis, eczema and dermatitis. This points the way for dietetic measures to reduce the predisposition while insulin treatment may benefit. Lortat-Jacob and Legrain have reported improvement from insulin treatment in patients with psoriasis when the blood cholesterol did not surpass 0.197 per cent, while it failed to relieve with 0.285 per cent. They have also called attention recently to the frequency of an inherited diabetic taint in patients with psoriasis. Hyperglycemia was found in fourteen of seventeen cases of psoriasis, and Lacroix has reported it in 25 per cent, and in 30 per cent of his cases of eczema, as also in 45 per cent of the cases of pruritus preceding diabetes or a dermatitis. On the other hand, the experiences related recently by Bernhardt and Zalewski did not confirm the conclusions as to the curative value of insulin.

DERMATOMYOSITIS. ACHILLE SEGA, Arch. di pat. e clin. med. **5**:213 (Aug.) 1926.

Two cases are described in detail, with the pathologic anatomy, representing the dermatomucomyositic type. The fever and the enlargement of the spleen testify to the infectious-toxic origin. Sega's patients were a youth, and a woman, aged 50. Both presented the characteristic intense dystrophia and mummified aspect of the soft parts; the disease ran its fatal course in eight and six months. The differential diagnosis is discussed.

CHANGES IN THE SKIN INDUCED BY IODINE. K. FRITZLER, Arch. f. exper. Path. u. Pharmakol. **114**:6 (July) 1926.

A given area of the skin of guinea-pigs was painted once with a given amount of tincture of iodine, and these areas were cut out for histologic examination at intervals, with controls. The acute inflammation which promptly developed was most intense on mucous membranes and least on the sole. It had never entirely subsided by the sixteenth day. Resorption from the skin depends on the site more, perhaps, than has been realized hitherto.

ACTION OF INTRADERMAL IRRITATION ON THE THRESHOLD FOR STIMULI. F. LUITHLEN and H. MOLITOR, Arch. f. exper. Path. u. Pharmakol. **114**:47 (July) 1926.

The wheal resulting from injection into the skin or cornea of physiologic sodium chloride solution enhanced the sensitivity to electric tests and also to the blood pressure raising-depressing influence of certain drugs that have a toxic action on the sympathetic or parasympathetic nervous system. The experiments on this threshold-lowering influence of a simple wheal were made on cats, decerebrate or under the influence of ether. The special drug tested was a cholin compound for the parasympathetic system; epinephrine for the sympathetic. The threshold-lowering influence was less constant with the sympathetic.

NEURODERMATITIS FROM OXYURIASIS: PATHOGENESIS OF PRURIGO. E. SCHRÖPL, Dermat. Wchnschr. **83**:1083-1097 (July 24) 1926.

In the four cases of chronic prurigo described, an inherited taint (alcohol) was evident, and all revealed rebelliously persisting oxyuriasis. Schröpl assumes that the mechanical and toxic irritation from the oxyurids affected directly the nerve centers in the region, the inferior dorsal and superior lumbar portions of the spinal cord. The changes in the local pain and temperature sense corresponded to this assumption, as also the similar aspect and localization of the prurigo in all, but the most convincing proof was the response to the roentgen rays applied to the region in one case, supplementing the focal reaction after intradermal injection of oxyuris extract. The itching always had become more intense under emotional stress. The roentgen rays were applied to this portion of the spine at three sittings, a strong erythema dose, a week apart. Benefit was apparent from the first, and the patient is now free from all symptoms of the disease from which he suffered for nine years. The interval since has been only three weeks, but even temporary arrest confirms the mechanism of the neurodermitis, Schröpl insists, as he explains it.

INHIBITION BY PROCAINE OF LIGHT DERMATITIS AND PIGMENTATION. S. ROTHMAN, Strahlentherapie **22**:729, 1926.

The wheal induced in Rothman's experiments by intradermal injection of a 1 per cent solution of procaine hydrochloride modified the skin so that it did not continue to share in the dermatitis and pigmentation induced by exposure to the mercury vapor lamp rays. No other substance tested possessed this property. The skin shared in the dermatitis evenly at first, but by the second, third or fourth day the area of the wheal blanched, the skin resuming its normal aspect, and this area showed no trace of the consecutive pigmentation. He has obtained this *Aussparungs* phenomenon, as he calls it, regularly in 100 subjects tested, and the contrast was almost as decided as at first by the sixth month in the two reexamined later. If the rays are so potent that blisters form, the procaine hydrochloride area is spared the vesicles. The procaine hydrochloride has no effect in this line if not injected until after the exposure. This confirms his assumption that it is responsible for an elective absorption of ultraviolet rays.

ABSORPTION OF ULTRAVIOLET RAYS IN HUMAN SKIN. W. SCHULTZE and S. ROTHMAN, Strahlentherapie **22**:736, 1926.

Continuing the research on absorption of rays by procaine, it was found that the absorption occurs under conditions which link this "sparing phenomenon" with the antirachitic action of light (Hess). It seems to involve the nonsaponifiable, acetone-soluble lipoids of the outer skin.

THE BIOLOGIC ACTION OF HARD AND SOFT ROENTGEN RAYS IN RELATION TO SKIN CANCER. W. ALBERTI, Strahlentherapie **23**:31-40, 1926.

The experiments were made on the cornea of the larva of the salamander, *Salamandra maculosa*, as the resulting changes in mitosis, etc., closely resemble those seen in malignant disease of human skin. The biologic action seemed to be independent practically of the quality of the rays, the results confirming Strangeways' and Wood's research on explants and cancer inoculations. There is first a stage of abnormal mitosis, then a stage of no mitosis; this is followed

by a phase of abnormal mitosis which differs however from that of the first stage. The facts observed justify, Alberti says, treatment of skin cancers with 200 kilovolts through a 0.2-0.5 mm. copper filter, exposing to 1 cm. on apparently sound skin beyond the lesion, and with 100 kilovolts through a 1-2 mm. aluminum filter for the cancer alone.

SPECIFIC SKIN REACTION IN LEPROSY. P. BARGEHR, Ztschr. f. Immunitätsforsch. u. exper. Therap. **47**:529-531, 1926.

Finely chopped tissue from nodules from a leprous patient was injected by the Pirquet technic in 100 other patients in all stages of leprosy. There was no reaction to the test in 82 per cent. A positive reaction was obtained in old chronic, stationary cases and in persons long living in contact with leprosy but without any manifestations of the disease. Subthreshold infections had evidently induced permanent immunity. Bargehr practices in Java. He adds that in active leprosy antibodies are not formed in sufficient amounts to conquer the disease.

ITCHING AS INITIAL SYMPTOM OF MAMMARY CANCER. J. H. VERHAVE, Nederl. Tijdschr. v. Geneesk. **2**:1082 (Sept. 4) 1926.

Küttner called attention in 1924 to two cases of mammary cancer in which intense local itching had been noted for months before the malignant disease became apparent. In a 1925 Amsterdam thesis, Sanders mentions the same phenomenon as noted in five of 125 cases of cancer of the breast in Noordenbos' service. In one in this group eczematous changes in the areola did not develop until a full year after the beginning of the itching in the skin. Verhave here reports a case personally observed in which the woman, aged 37, had been having itching in the skin of the right breast for months. It finally became so severe that she applied for treatment; there had been no pain at any time, and the patient was well nourished and had suckled her one child. The breasts were pendulous, of normal aspect otherwise, but palpation disclosed the tumor in the lower outer quadrant.

THE OCCURRENCE OF THE STREPTOCOCCUS ON THE HEALTHY AND THE DISEASED SKIN. H. HAXTHAUSEN, Hospitalstid. **69**:637 and 670, 1926.

With crystal violet the streptococcus grows unhindered in fluid and on solid blood agar mediums while the growth of the staphylococcus is checked. With crystal violet bouillon the results are not positive quite so often as with Sabouraud's pipet technic, but on blood agar the new crystal violet method has the advantage of allowing estimation of the numbers of the streptococci, and thus it effectually supplements the pipet technic. Six hundred tests were made with the crystal violet on 400 subjects, culturing scraps of skin in broth and on blood agar. Streptococci were found in skin taken from various regions in seven of ninety-one healthy persons. They were likewise present in 15 per cent of 200 subjects tested who had forms of skin disease (twenty-seven varieties) for which the streptococcus is not supposed to be responsible. On the other hand, the results in the other subjects with skin diseases for which the streptococcus has been held responsible confirm the assumption that it should be incriminated for impetigo, ecthyma, intertrigo and pityriasis of the face. The experiences related indicate that Jadassohn's form of impetigo is essentially different from common impetigo, and that not all the forms of

intertrigo are due to the streptococcus. A further outcome of the research reported is that the streptococcus seems to have a hitherto unsuspected causal importance in other forms of pityriasis besides the acute "epidemic" facial variety.

SERODIAGNOSIS OF FRAMBESIA AND SYPHILIS. M. HASHIKUCHI, Acta derm. (Japan) **7**:749, 1926.

An alcoholic extract of pig heart, with cholesterol, is proving a useful antigen in the serodiagnosis of syphilis and yaws. Comparison of this precipitation reaction and the Wassermann reaction in frambesia and in syphilis showed concordance in 96.7 and 94.5 per cent of the cases. Comparison with the Meinicke test in different stages of frambesia showed concordance in the second and latent periods, while Hashikuchi's antigen yielded more positive results in the first and third stages (from 91.6 to 87.5 per cent in the first stage). Even from the serologic standpoint it is difficult to distinguish between syphilis and frambesia.

IMMUNOLOGIC RELATIONS BETWEEN SYPHILIS AND FRAMBESIA. S. MATSUMOTO, Y. IKEGAMI and S. TAKASAKI, Acta derm. (Japan) **7**:753, 1926.

If rabbits are again inoculated with syphilis within a certain period after the primary inoculation, infection does not occur, but these rabbits can be easily infected with frambesia virus. The inoculation induces the typical frambesia lesions at the point of entrance. This susceptibility to yaws with the resistance to superinfection with syphilis testifies to the difference between the two diseases.

CUSHING, Chicago.

DERMATOLOGIC ABSTRACTS

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

SCLEREMA NEONATORUM: CHEMISTRY OF SUBCUTANEOUS TISSUES: II. G. A. HARRISON, Arch. Dis. Childhood **1**:123 (June) 1926.

Five cases of sclerema neonatorum (adiposum) are reported by Harrison, with postmortem and histologic examinations in four. The fifth patient recovered. In two cases there were cysts in the subcutaneous fat. Histologically, in the four cases, the subcutaneous fat showed evidence of chronic inflammation and repair, with giant cell formation. In each case there were abundant anisotropic crystals in the fat and lying free. The roentgen ray revealed opaque deposits. The chemistry of the subcutaneous fat has been studied in four cases—extensively in two. The melting point was raised slightly, and there was an increase in the total cholesterol, but the iodine and saponification values were similar to control cases of corresponding ages. The view that sclerema is a primary inflammation of the subcutaneous tissues of unknown cause is upheld.

MALARIA IN TREATMENT OF GENERAL PARALYSIS. R. F. L. RIDGWAY and E. M. GREEN, Atlantic M. J. **29**:544 (May) 1926.

Ridgway and Green report thirty-four patients suffering from general paralysis who were treated by inoculation with blood from one having malaria of the tertian type. Twenty-six of these patients recovered from the inoculated

disease; seven died, and one failed to become infected. Of the twenty-six patients, five were not benefited by the treatment; nine were improved in greater or less degree, and twelve exhibited complete remissions. Seventeen of the number have been paroled from the hospital, and sixteen are still at home, many of them having reengaged in their former occupations. The results obtained in the series of cases prove that there may be no correspondence between the mental improvement and the changes in the physical signs of the disease nor in the serologic picture. Several of those manifesting complete remissions exhibited no favorable modifications in these respects. In only a small proportion of the cases did the Wassermann tests of the blood and the spinal fluid show changes which could be attributed to the malarial process. While physical signs were favorably modified in a number of instances, in no case did they all show a return to the normal.

TREATMENT OF EARLY SYPHILIS. J. E. MOORE and A. KEIDEL, Bull. Johns Hopkins Hosp. **39**:1 (July) 1926.

Moore and Keidel present a plan of treatment for early syphilis, the essential features of which are (*a*) that treatment shall be continuous, consisting of courses of arsphenamine alternating with courses of mercury by inunction (or of insoluble bismuth salts intramuscularly) plus potassium iodide; (*b*) that treatment shall be carried out under detailed serologic control; (*c*) that treatment shall be prolonged without intermission for one year after the blood and spinal fluid have become and have remained completely normal.

TREATMENT OF EARLY SYPHILIS: II. CLINICAL RESULTS. J. E. MOORE and J. E. KEMP, Bull. Johns Hopkins Hosp. **39**:16 (July) 1926.

Moore and Kemp report the clinical results of treatment in 402 patients with early syphilis, of whom forty-two had seronegative primary syphilis, sixty-one seropositive primary syphilis and 299 early secondary syphilis. Under a system of continuous treatment with alternating courses of arsphenamine and mercury, the probability of cure is in direct proportion to the duration and regularity of treatment. Only 10 per cent of those patients receiving eight or less doses of arsphenamine without mercury were cured; when two courses of arsphenamine, plus interim mercury, were given, 37 per cent were cured; after three courses, 56 per cent, and after four or more courses, 78.8 per cent of probable cures were obtained. The optimal amount of treatment for early syphilis with the plan advocated appears to be a full year of treatment after the serology of blood and spinal fluid has become and has remained negative, excepting only in seronegative primary syphilis, in which a three course treatment, lasting nine months, apparently produces satisfactory results. With the utmost cooperation on the part of the patient, and painstaking treatment and follow-up on the part of the physician, it appears possible to cure 100 per cent of patients with seronegative primary syphilis, but only 80 to 95 per cent of those with seropositive primary or early secondary syphilis. The tendency of inadequately treated syphilis to repeated relapses in the same patient is pointed out. Cardiovascular syphilis, especially aortitis, was the most common type of tertiary recurrence observed. The evidence of repeated routine spinal punctures in fifty-four patients permits the general statement that in early syphilis, if the spinal fluid is normal after six months' treatment, the patient will not develop clinical neurosyphilis or a subsequently positive spinal fluid unless a second dissemination of organisms (clinically manifest by recurrent secondary syphilis

or a recurrent positive blood Wassermann reaction) occurs. Periodic reexaminations from physical and serologic standpoints are absolute essentials in the evaluation of treatment results.

THE INCREASE OF SYPHILIS. PARIS LETTER, J. A. M. A. **87**:1048 (Sept. 25) 1926.

After the war, syphilis decreased in France, as a result of the crusade that was waged against it. The widespread incidence of the disease was due, in part, to the foreign soldiers in French territory and to relaxation of morals that is associated with troubled periods. The American army medical corps organized rigorous prophylaxis and treatment of venereal diseases, and American troops presented the best condition in this regard.

The war and the example of the foreign sanitary service on French soil helped to remove the traditional reticence of the French toward public mention of venereal disease. Public dispensaries sprang up in all parts of Paris, whereas formerly the city had had only two public consultation centers for venereal diseases, one in the Hôpital St. Louis and one in the Hôpital Ricord. Thus combated, syphilis soon decreased rapidly. Furthermore, the decrease has been general throughout Europe wherever the antivenereal crusade was seriously undertaken. In Geneva, the two Red Cross dispensaries encountered last year only one syphilitic chancre. In Belgium, according to Dr. Schroenen, secretary of the Ligue nationale belge contre le péril vénérien, the cases of primary syphilis have decreased 75 per cent. Also in Denmark and Spain the diminution has been continuous. In Vienna, Professor Finger states, the decrease, since 1919, has been 44.6 per cent. Russia seems to be the only country in which syphilis has increased since the war, although the Bolshevists state that the increasing number of chancres treated in the dispensaries is due to the fact that the people are becoming more enlightened and are more ready to consult a physician than formerly.

In France, from 1919 to 1923, syphilis continued to decrease. In 1922, however, the recedence of the disease was less marked. In 1924, the incidence remained practically stationary. Since the middle of 1925, it has again begun to increase. M. Jeanselme, professor of dermatology and syphilology at the Faculty of Medicine of Paris, has conducted an inquiry in Paris and in the large provincial cities, to discover the causes of the increase. He reached the conclusion that the two main causes are (1) the increase of foreign laborers, and (2) the abandonment, by many physicians, of intravenous injections of arsphenamine for newer remedies of less efficacy.

As a result of the heavy losses of the war (greater in France than in any of the allied countries), the diminution of the birth rate, and the scarcity of man power, especially males, there are today three million foreign workers in France: Poles, Czechoslovakians, Jugoslavs, Italians, Spaniards and Algerians. They are a manifest cause of the spread of contagious disease, not so much by reason of the diseases that they bring in as that, because of the fact that they are grouped together in small colonies, they escape, more than the French workers, sanitary supervision. These colonies are hotbeds of prostitution. Then again, those infected do not seek treatment so long as their strength keeps up, and the number of such is very great to judge by the number of them who finally seek admission to the hospitals, where they constitute from 10 to 20 per cent of the inmates. Such persons are for the most part unmarried clients of prostitution and remain infective for long periods. In his service in the Hôpital St. Louis, Professor Jeanselme reported that 20 per

cent of the patients were foreign syphilitic patients. In the free dispensaries, the most recent statistics record, among 451 cases of recent syphilis, 260 French patients, 140 foreign patients, and fifty-one natives of northern Africa. The inquiry showed that in 18 per cent of the cases syphilis had been brought in from foreign countries and that in 82 per cent it had been contracted in France, but almost always in the colonies of foreign workers.

The same conditions have been observed in the provinces, especially in the seaports of Marseilles and Havre and in Rouen, where foreign workers congregate in large numbers and where syphilis is continually brought in from without. At Bordeaux, Dr. Pettges states that 33 per cent of the syphilitic patients are foreigners. In the region of Nancy, where there are large numbers of Italians, Poles and Czechoslovakians, the proportion is similar. Dr. Laurent has made the same observations in the vicinity of St. Etienne.

In Belgium also there is a great influx of foreign man power, and syphilis persists although it is on the decrease. Here Professor Jeanselme, together with other physicians of the Ecole de St. Louis, incriminates another cause which is more particularly applicable to France; namely, the inadequate character of the treatment, since physicians show a tendency to abandon intravenous injections of arsphenamine. The discovery of the antisyphilitic properties of bismuth salts by Sazerac and Levaditi has played a part. Jeanselme states that since, in 1921, the investigations of Fournier and de Guérot confirmed the value of bismuth salts, syphilis ceased to retrogress. M. Jeanselme and M. Milian admit that bismuth is useful, particularly in the treatment of neurosyphilis or as supplementary treatment when there is an intolerance for mercury or arsenicals; but they assert that it should not take the place of these in treatment of primary syphilis or when it is desired to bring about the eradication of syphilis. The suppression of the local manifestations from which contagion results depends on the rapid action of these two remedies.

MINERAL CONTENT OF HUMAN, DOG AND RABBIT SKIN. H. BROWN, J. Biol. Chem. **68**:729 (June) 1926.

Data are presented by Brown giving the results of determination of calcium, magnesium, sodium and potassium in the skin of man, the dog and the rabbit. Individual variability in mineral content in the same species as well as distinct differences among different species are pointed out.

MELANOMA OF EYE IN SYPHILITIC RABBIT. W. H. BROWN and L. PEARCE, J. Exper. Med. **43**:807 (June 1) 1926.

Brown and Pearce noted the development of a melanotic tumor in a defective eye of a syphilitic rabbit following repeated genital inoculations with *Spirochaeta pallida*. The appearance of the tumor coincided with the development of a syphilitic lesion in the same eye, suggesting a relation between the occurrence of the two lesions.

TRYPARSAMIDE IN TREATMENT OF LATE NEUROSYPHILIS. J. R. MOORE and I. C. SUTTON, J. Nerv. & Ment. Dis. **63**:569 (June) 1926.

In the selection of patients for this treatment, it is the impression of Moore and Sutton that those who have received prolonged and intensive saturation with mercury and the arsphenamines show more striking gains than those who have

received no preparatory treatment. The previously treated patient is also less subject to the Herxheimer-like flare-ups so often seen during the first course of tryparsamide injections. It is emphasized that tryparsamide is primarily a resistance builder and a healer of lesions, but not a spirocheticide. This fact precludes its use in early syphilis. It greatest worth is in general paralysis and late tabes dorsalis, and in selected cases of central nervous system syphilis which have resisted systematic and combined intravenous and intraspinal therapy with the arsphenamines.

HERPES ZOSTER OF BLADDER. F. E. DUBOIS, J. Urol. **15**:583 (June) 1926.

The diagnosis in Dubois' case was based on the fact that cystoscopy showed typical herpetic vesicles coincident with a typical herpes zoster of the buttock of the same side. It is of note that the nerve supply of the buttock and of the base of the bladder is the same; i. e., from the sacral plexus. The diagnosis was further confirmed by the more or less rapid disappearance of both the cutaneous and the mucosal lesions at the same time, without any particular treatment, other than mild urinary antiseptics and a local application of methylene blue to the external lesions.

COMMON WARTS OF MOUTH. W. D. DAVIS, Missouri State M. A. J. **23**:144 (April) 1926.

Of the two cases presented by Davis, one was a single verruca of the tongue; the other patient had an enormous number of verrucae in the mouth.

VARICELLA ENCEPHALITIS AND VACCINIA ENCEPHALITIS. D. W. WINNICOTT and N. GIBBS, Brit. J. Child. Dis. **23**:107 (April-June) 1926.

A case of varicella encephalitis is reported by Winnicott and Gibbs and twelve other cases which have been described in the last thirty years are quoted or abstracted. A case of vaccinia encephalitis is also described, and forty-four other cases of possible vaccinia encephalitis are quoted. Two cases of measles encephalitis are described, and brief reference is made to the published cases.

VACCINE THERAPY OF ACRODYNIA. A. E. VIPOND, Brit. J. Child. Dis. **23**:127 (April-June) 1926.

In the treatment of acrodynia patients, Vipond claims universal success from the use of a vaccine made from a diplococcus which he recovered from an inguinal node. He gives each patient 1 cc. of the vaccine subcutaneously as a primary dose. In three days the patients show a marked improvement. He then gives from 1.5 to 2 cc. as a dose. After from three to four doses more of 4 or 5 cc. of the vaccine, the patients are practically cured.

CASE OF HERPES RECURRENS. C. I. WRIGHT, Brit. J. Child. Dis. **23**:132 (April-June) 1926.

Wright reports the case of a girl, aged 12, who following a moderate attack of diphtheria, developed an herpetic eruption on both cheeks. Its distribution was asymmetrical, being over the subzygomatic and midmandibular regions on the left, and over the malar eminence on the right. Two days later several rounded, tense unilocular bullae were present on both sides. Culture of the

contained fluid was sterile. No source of reflex irritation was apparent other than the recent throat condition. For the last three years the patient has had recurrent attacks of herpes on one or other cheek about three times a year, usually associated with the onset of a cold. In no previous attack had the lesions been bilateral. There was no personal or familial history of urticaria, asthma, migraine, gout, malaria, syphilis or tuberculosis.

Case of Death Due to Bismuth Injection. C. F. Chenoy, Indian M. Gaz. **61**:234 (May) 1926.

Two hours after receiving an intramuscular injection of a bismuth compound into the buttock, Chenoy's patient died with every evidence of bismuth poisoning. Chenoy says that the man's physician must have inadvertently injected the bismuth into a vein.

Xanthoma Versus Gout. A. Gilbert et al., Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1067 (June 18) 1926.

Gilbert, Chabrol and Bénard's patient, aged 18, presented multiple tophi beneath the skin of the limbs, also intradermal xanthomas profusely disseminated over the body. The tophi first appeared at the age of 4. The number of them gradually increased without causing any pain. The young man did not present any signs of gout or rheumatism. In roentgenograms, the semilunar cartilages and bones seemed to be intact. The nodules proved to be composed exclusively of cholesterol. Blood examination showed a considerable increase of cholesterol. The coexisting hypoglycemia may explain the failure of insulin treatment, continued for about ten days, in this case. Investigation revealed the presence of xanthoma growths in four generations of the family. In fourteen members of the fourth generation, the xanthoma patches started at the age of 4.

Sclero-Edema in the New-Born. P. Woringer, Rev. franç. de Pédiat. **2**:63 (April) 1926.

Woringer has observed within the last three years six cases of sclero-edema of the suprapubic region, sometimes spreading to the external genitals, abdominal wall or thighs. The infants were all boys, born before term. The affection lasted from four weeks to five months. The prognosis is good; cure occurred without any treatment. No connection between the edema and an infection could be established; suppuration of the umbilical wound was noted in only one case, infection of the genital organs in none. No signs of syphilis could be detected. Thus the pathogenesis of the edema is still obscure. It is elastic, but not enough to retain the imprint of the finger.

Sanocrysin Exanthem. Bonorino Udaondo and Vadone, Presna méd. argent. **13**:1 (June 10) 1926.

In the case illustrated, a man with pulmonary tuberculosis and no previous skin affection was being treated with sanocrysin when he developed intense gingivitis and stomatitis, with an extensive eruption, predominating on the hands and arms. Similar manifestations have been noted under treatment with other gold salts, and others have reported eruptions in a certain proportion of their sanocrysin-treated cases.

CLIMATIC DERMATITIS. E. ESCOMEL, Gac. méd. de Caracas **33**:131 (May 15) 1926.

Escomel describes a dermatitis which is common in Peru when persons living in the mountainous regions move to a lower, drier level. It affects those with a delicate skin, a papular vesicular erythema which is not contagious, and may yield to the usual treatment for urticaria. It develops within one or two days or by the fourth day, and the itching is intense. It subsides promptly on return to a higher altitude, but otherwise may heal spontaneously or long persist and recur. The local name for it is chapetonada, as attention was called to it by the number of Spaniards attacked by it in their conquest of Peru, and the Spaniards were called then chapetones. In the rare rebellious cases, autohemotherapy has proved a valuable aid.

KIDNEY TUMOR WITH CONGENITAL SYPHILIS. R. RINALDI, Arch. ital. di urol. **2**:582 (May) 1926.

Rinaldi states that the nephrectomy was borne by the girl, aged 16, whose adenosarcoma of embryonal type had developed rapidly. The kidney vessels and connective tissue showed also chronic lesions of inflammatory nature from the congenital syphilis.

THE SKIN IN CHILDREN. J. V. PAJARES, Pediatria españ. **15**:125 (May) 1926.

Velasco Pajares remarks that the natural complexity of dermatology is magnified in children as special tendencies modify the typical manifestations in the skin. Pallor, angioneurotic conditions, dryness, hyperhidrosis and seborrhea are the reflection of various internal abnormalities. In one child, aged 20 months, with congenital syphilis, the skin had a cottony aspect and consistency. It was extremely white and lusterless, and readily taken up in folds, as if it were too large for the tissues beneath. This cottony skin he has always found associated with the thymicolymphatic constitution, and in the case of this child he warned the parents and others present at the examination that sudden death might follow even some very slight maneuver. He then proceeded to introduce the tongue depressor to examine the pharynx for the congenital stridor. Hardly had the depressor been introduced when the child died suddenly, and all attempts at resuscitation failed. This white cottony skin is a warning of thymicolymphatic peril. The angioneurotic skin seems to be associated with spasmophilia; hyperhidrosis with rickets; a greasy skin with the exudative diathesis; a loose skin with tendency to phlyctenas.

ACRODYNIA. J. C. RECALDE CUESTAS, Rev. méd. del Rosario **16**:75 (May) 1926.

Recalde Cuestas relates that dengue appeared at Rosario in 1925, the first recorded instance of its prevalence. At the same time he encountered eight cases of acrodynia or "pink disease" in children, aged from 6 weeks to 3 years. The clinical picture included fever for three days, with sudden decline as an eruption appeared. There was no desquamation. The blood count was characteristic: extreme leukopenia, with considerable reduction in the numbers of polymorphonuclears, and high relative mononucleosis. Recovery was prompt and complete. An erythematous pharyngitis was constant, and one child presented intense otalgia and another extreme general hyperesthesia.

LOCALIZATION OF HEMATOGENOUS SKIN INFECTIONS. F. KOGOJ, Arch. f. Dermat. u. Syph. **150**:333 (May 22) 1926.

Kogoj found that injection of internal organs of guinea-pigs and rabbits with a culture of the fungus of mouse favus was followed in most cases by hematogenous foci in unirritated as well as in irritated portions of the skin, but that the foci appeared with greater frequency in the irritated portions. Up to a certain point, it was possible for the experimenter by applying strong irritation to elect the site of the skin lesion.

SKIN LESIONS AND CUTANEOUS GLAND SECRETIONS. H. FISCHER, Dermat. Wchnschr. **82**:805 (June 12) 1926.

Fischer found that certain changes in many diseases affecting chiefly the epidermis were paralleled in the local irritation set up by excretion through the cutaneous glands of parenterally administered chemicals.

SYPHILITIC INFECTION. W. KOLLE and E. EVERS, Deutsche med. Wchnschr. **52**:1075 (June 25) 1926.

Kolle and Evers found spirochetes in inguinal lymph nodes of rabbits in amounts sufficient for infection of other rabbits as soon as thirty minutes after rubbing the infectious material for one minute into the scratched skin of the scrotum. The distance from the point of infection to the lymph nodes was about 6 cm. Similar experiments on guinea-pigs revealed spirochetes in the lymph nodes as early as five minutes after infection. Recurrent spirochetes and trypanosomes invade the lymph glands within two minutes. It is therefore evident that any attempt at local disinfection after the intercourse has very little chance of success. It is better to administer a strong dose of arsphenamine intravenously.

ACANTHOSIS NIGRICANS AND CANCER. H. KÜTTNER, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **39**:276, 1926.

Küttner does not regard acanthosis as an absolute indication for exploratory laparotomy, since a tumor was found in only 45 per cent of the cases. If the patient is young and suffers from familial obesity or diabetes, or other disturbances of development, the acanthosis may be idiopathic. Laparotomy should be considered seriously in every other case. Acanthosis is characterized mainly by a pigmentation and papillary condition of the skin.

EXPERIMENTAL IDIOSYNCRASY TO NICKEL. B. WALTHARD, Schweiz. med. Wchnschr. **56**:603 (June 19) 1926.

Since practically every worker occupied with nickel plating suffers sooner or later from eczema, Walthard selected nickel salts for experimental production of idiosyncrasy in guinea-pigs. He succeeded, although the animals showed considerable individual differences. When he reapplied nickel salts after the first eczema had healed, the reaction occurred much quicker than after the first application, even when a different part of the skin was used. The eczema flared up temporarily in some of the animals after subcutaneous injections of 1 cc. of a 0.1 per cent solution of nickel sulphate at a part remote from the eczema. Intravenous injections induced no shock, but a moderate shock was observed after subcutaneous injections in animals that had nickel eczema. This shock was followed by rapid recovery from the eczema in spite of continued application of the salt. Skin tests failed.

Society Transactions

MINNESOTA DERMATOLOGICAL SOCIETY

Regular Meeting, Feb. 3, 1926

PAUL O'LEARY, M.D., *President, in the Chair*

MACULAR ATROPHY. Presented by DR. SWEITZER.

A woman, aged 24, stated that about three years before presentation she had noticed peculiar lesions of the skin. The first lesion was on the forehead and others have since appeared on the face and upper part of the chest, front and back. Numerous small white areas were present. The skin showed wrinkling and ballooning, and the area felt depressed to the examining finger.

DISCUSSION

DR. SWEITZER: This is the second one of a general group presented before this society. It is rare. I think macular atrophy, stria atrophicans and some types of scleroderma are closely allied. This case is similar to one Dr. Michelson had except that there is not such a rigid edge to the lesion; this has only the flat atrophy without so much loss of tissue. Dr. Michelson made a good biopsy examination in this case, and some of the histologic characteristics resemble those of scleroderma.

DR. MICHELSON: All the conditions which are grouped under macular atrophy of the skin, and especially circumscribed atrophy, are interesting. Although the case presented tonight resembles my case of Schwenniger-Buzzi tumor-like growths, the bulging is not so marked. Nevertheless, I think the conditions are identical, both being macular atrophies, and the trouble probably began in the corium, the epidermis being involved secondarily. In this case the elastic fibers appeared only as little fragments, and the epithelium consisted of only two or three layers of cells. In the section I examined there were no areas of infiltrate, the condition being well along toward the end stage. Macular atrophy may show different degrees of pigmentation; in some of the lesions there is a yellow or a xanthomatous color. In others the lesions may assume a slatelike hue; while again in the morphea type a lilac halo may surround the lesion.

DR. BUTLER: Would not von Recklinghausen's disease give a similar picture?

DR. MICHELSON: The pigmentation in von Recklinghausen's disease is more lentiginous or brown.

DR. SWEITZER: Dr. Fred Wise wrote of von Recklinghausen's disease without tumors but with pigmentation.

DR. MICHELSON: I am willing to grant von Recklinghausen's disease without tumors, but I think pigmentation is always present.

DR. O'LEARY: I think the pigmentation is one of the diagnostic points.

SCALING SERPIGINOUS SYPHILID OF THE PALMS AND SOLES. Presented by DR. SWEITZER.

A girl, aged 15, was first seen in December, 1925. At that time she presented a thickening of both palms, the condition in some places running up on the wrists and backs of the hands. The edges were regular and sharply defined, and the center of the area involved showed motheaten pitlike depressions similar to those seen in palmar syphilis. The soles showed a serpiginous, papular eruption with no evidence of vesication. The Wassermann reaction was negative, and examination was negative for fungus, so the diagnosis was reserved and a therapeutic test instituted. She was given bismuth hypodermically and improved rapidly. In view of this rapid improvement without any local therapy, a diagnosis of syphilis was made.

DISCUSSION

DR. SWEITZER: The patient's condition is vastly different now than when first seen. The possibility of psoriasis, ringworm, eczema and syphilis was considered. No fungus had been found. The lesions on the feet looked more like serpiginous syphilid. The lesions on the palms were sharply outlined, hyperkeratotic and motheaten, and looked like syphilis. The patient, however, had a negative Wassermann reaction and no stigma of syphilis, so I reserved the diagnosis. After ten injections of bismuth she has improved to her present condition.

DR. BUTLER: I do not think this is syphilis, but a fungus condition. It usually is not difficult to recover the fungus from the lesions on the foot, but it is more difficult to recover them from the palmar lesions. The girl gives a history of sweating of the feet, and in epidermophyton lesions there is a history of profuse sweating.

DR. FREEMAN: I do not think the lesion is syphilitic. She has had no other treatment and no history of any other eruption, and with the negative Wassermann reaction I would rule out syphilis.

DR. IRVINE: I would not consider syphilis in view of all the negative factors.

DR. OLSON: The patient probably has the hyperkeratotic variety of epidermophytosis. There was one large vesicle on the sole of the foot. The fungus is readily found in the vesicular type of the disease on the foot, but is found with difficulty from the hyperkeratotic lesions.

DR. O'LEARY: I am rather more inclined to a diagnosis of syphilis than of fungus infection. With the definite improvement after bismuth medication, definite vesicles and scaling that disappeared rapidly, I am a little inclined to say syphilis.

DR. MICHELSON: I cannot offer a definite diagnosis, but the question is, where should one stop treatment with this patient. One would not be justified in putting her under a long-continued antisyphilitic course of treatment with the evidence at hand.

DR. O'LEARY: I should like suggestions as to treatment.

DR. BUTLER: If you will remove a plug of this hyperkeratotic tissue, and make very thin sections of it, I think you will find the fungus, although it may take a little time.

DR. SWEITZER: No vesicles have been found on the feet; only dark papules, grouped and serpiginous.

RETENTION CYST OF LOWER LIP. Presented by DR. SWEITZER.

PYOGENIC GRANULOMA. Presented by DR. SWEITZER.

C. C., a man, aged 57, was presented with a papillary growth on the forehead. This growth followed an injury to the forehead two weeks before.

DISCUSSION

DR. SWEITZER: I have usually used the cautery for this particular type, and I think it will take this off rapidly.

DR. MICHELSON: I sent a reprint of my recent article on pyogenic granuloma to Professor Kren, who, in acknowledging its receipt, stated that he had seen only one case in the last seven years in Vienna. I think this is worth recording, because we see them constantly.

D. D. TURNICLIFF, M.D., Secretary.

Regular Meeting, April 7, 1926

PAUL B. COOK, M.D., *Vice President, in the Chair*

MELANOMA OF THE ARM. Presented by DR. BOREEN.

L. R., aged 25, a university student, presented an ulcerated lesion of the upper left arm. The ulcer was shallow, about one-half inch in diameter, and surrounded by a deep purplish pigmentation. In the pigmented area was a small pea-sized nodule. There were no other lesions and no palpable glands. The lesion had been present for ten months.

[NOTE.—Later the lesion was excised by wide excision and biopsy examination showed typical malignant melanoma.]

A CASE FOR DIAGNOSIS. Presented by DR. SWEITZER.

Mrs. McC., aged 36, complained of an eruption on the sides of the thorax. The eruption had occurred at menstrual periods for three months. At the time of presentation the eruption showed a diffuse dermatitis on the sides of the thorax and on both legs.

DISCUSSION

DR. SWEITZER: This patient came in several months ago with an acute dermatitis which was papular, with irregular edges, and slightly red. Then she disappeared from observation. She came in the other day, and when I asked her why she had not come back, she said the eruption had all gone away. It had come on with menstruation. I do not think the eruption is a dermatitis dysmenorrheica, but I am going to keep it under observation.

COLLOID DEGENERATION OF THE SKIN. Presented by DR. BUTLER.

A woman, aged 26, married, had developed pinhead to hemp-seed sized, yellow, translucent nodules on the upper part of the cheeks five years before. The nodules were flat and resembled vesicles or might be mistaken for xanthoma lesions. The nodules were discrete but closely aggregated, and after attaining a certain size became stationary. None of them had involuted. There was slight pigmentation in the affected area. There were no subjective symptoms.

LICHEN PLANUS. Presented by DR. SHAPIRO.

Mrs. S. S., presented lesions on the arms, trunk and legs, mostly on the flexor surfaces. The lesions consisted of papules which were markedly raised; they were arranged in linear grouping, sharply demarcated one from the other, were violet red, itched severely and came on after a nervous experience this woman had following the death of her daughter-in-law. The patient was improving on treatment with bismuth.

ACNE ROSACEA. Presented by DR. SHAPIRO.

LICHEN SPINULOSUS. Presented by DR. SWEITZER.

E. S., a girl, aged 14, presented small patches of spicules on the back and the arms, of about four months' duration.

ICHTHYOSIS HYSTRIX. Presented by DR. SHAPIRO.

L. K., aged 7½ years, presented lesions on the scalp, neck, chest, back and arms. These lesions had been present since birth. There was a marked thinness of the hair in certain places, with a marked dry scaly condition. The scalp was dry and rough, and the hairs also dry, coarse and rough. The typical lesion appeared on the neck. There were groups of pointed excrescences, mostly linear in arrangement. These groups comprised numerous pointed papules which were course, rough and dry to the touch and dark brownish in appearance, making it look as though the skin was dirty in these places. There was no scaling to these lesions. There was no symmetry. On the trunk the lesions appeared dirty and the areas involved were whitish in appearance, suggesting a scarring with the skin sunken below the general level, and they felt rather smooth. The rest of the body showed a general dryness and roughness.

NEUROFIBROMATOSIS. Presented by DR. BUTLER.

Miss E., aged 23, on January 20 had chills and fever, with swollen and painful cervical glands. A few weeks later a subcutaneous tumor the size of a pigeon egg was discovered by palpation in the left popliteal region of both legs. Later examinations from time to time revealed the presence of numerous subcutaneous tumors (a dozen or more) in different parts of the body. The tumors were located under the subcutaneous fat, and the skin over the tumors was not raised or changed in color. The patient had a brown pigmentation on the body that had existed since birth. Microscopic examination showed firm connective tissue stroma of new fibers of collagen with mucoid substance and oval or spindle shaped cells.

SCLERODERMA. Presented by DR. SWEITZER.

Miss G., aged 32, noticed a hard spot on the arm about one year before presentation. This spread slowly over the arm and up on the right shoulder. The patient showed a white irregular hard lesion on the right arm and shoulder.

EPITHELIOMA. Presented by DR. SHAPIRO.

Mr. B. W., a widower, aged 74, showed a lesion on the right ear of three years' duration. It was located on about the middle of the ear on the anterior surface, pretty well toward the edge, and was approximately square in its area, the

diameter being about 1 inch (2.5 cm.) in each direction. The skin over the lesion was gone entirely, leaving a raw surface. At first the edges were a little hard and infiltrated. The patient had had applications of radium at three different times and after each application the lesion would heal and be almost entirely gone, and then break down again. There were no gland conditions in the neck.

EPITHELIOMA OF THE MOUTH. Presented by DR. SHAPIRO.

T. O. L., a man, aged 56, presented an epithelioma of three months' duration. There were two lesions, one on the tongue and the other running down the inside of the cheek. The lesion of the tongue was underneath on the right side, more than half way back. It was a hard infiltrated mass measuring about 1 inch in diameter and ½ inch the other way. It was glistening rose red, and we could see a few scattered gray white patches indicating a beginning ulceration. This was sharply outlined. It did not bleed readily.

The other mass was extensive and seemed like a pica running from the palate outward and latterly down over the cheek toward the gum. It was soft, bleeding readily. There was not much pain here.

There were enlarged glands on both sides of the neck, especially on the right side.

DISCUSSION

DR. SHAPIRO: I have used radium three times on the epithelioma of the ear, and the lesion heals but promptly breaks down.

The other patient undoubtedly has a squamous cell carcinoma, but I did not wish to make sections until the operation has been performed.

DR. BUTLER: In epithelioma which involves the cartilage the cautery should be used, as the treatment must necessarily be destructive.

DR. SWEITZER: Regarding mouth cases, the cautery is not efficient in carcinoma of the tongue. If possible, I would send to Chicago for some radium emanation points and stick them in the tongue.

PARAPSORIASIS. Presented by DR. TURNACLIFF.

A man, aged 68, presented numerous discrete fawn-colored flat areas varying in size from 1 cm. to 5 cm. across, and located on the arms, body and legs. These lesions had a slight scale and were pruritic.

DISCUSSION

DR. TURNACLIFF: The extreme itching is the only odd feature about this case I believe.

DR. BUTLER: This man is an old patient at the clinic. The lesions correspond to parapsoriasis en plaque but I have never yet known of such lesions itching. It is suggestive of premycocic fungoides.

DR. BOREEN: I agree with the diagnosis of parapsoriasis in this case. Perhaps the itching might have something to do with the patient's age, i. e., a senile pruritis, but the lesions are typical of parapsoriasis.

DR. SWEITZER: I presented the case of an old lady several years ago in which I finally made a diagnosis of parapsoriasis. I always had a suspicion that the diagnosis in that case would have to be changed. I believe that if you keep this case under observation you will find that some change will take place.

SECONDARY SYPHILIS. Presented by DR. TURNACLIFF.

ALOPECIA. Presented by DR. COOK.

A woman, aged 35, showed a slowly progressive ring of alopecia at the junction of the hairy scalp and the forehead. At the end of about six months' duration this was about 6 inches (15 cm.) in length and 1½ inches (4 cm.) wide. The Wassermann reaction was negative, and the general examination gave negative results. There was no scarring in the area of the alopecia.

DISCUSSION

DR. COOK: I never saw alopecia in that location, slightly progressive, and extending over a period of six months, without some definite reason for it. It does not look like alopecia areata, but that was the only diagnosis I could make in the case.

ERYTHEMA INDURATUM. Presented by DR. MICHELSON.

ERYTHEMA INDURATUM. Presented by DR. BUTLER.

Miss M. A., aged 18, showed erythematous subcutaneous nodules which were from pinkish red to blue, of two years' duration. There was ulceration on both legs, with scarring. The patient had returned with a relapse of the condition, and showed tender subcutaneous tumors, and swollen cervical glands.

DISCUSSION

DR. SWEITZER: I had one patient, aged 25, who had this condition for three years; otherwise she was in good health. There were several lesions and scars on the legs. She gave a history of the breaking out occurring in the spring and the condition lasting until fall. In most of the cases I have seen the breaking out occurred in the winter. I have tried treatment with the lamp on some patients, roentgen ray and arsenic, but they did not show much improvement.

I was somewhat of the opinion that Dr. Michelson's case might be more inflammatory than erythema induratum. Erythema induratum usually breaks down. One patient I treated with arsphenamine did not improve; it is not a cure-all by any means.

DR. BUTLER: It depends a good deal on the type. There are two types of erythema induratum. One is distinctly nontuberculous, and corresponds to Dr. Michelson's case; and the other shows tuberculous structure. Of course, the therapy is different in those cases. The case I presented is of the tuberculous type.

DR. GAGER: Erythema syphiliticum is another type. Arsphenamine gives good results in those cases.

A CASE FOR DIAGNOSIS. Presented by DR. BUTLER.

A man, presented a lesion on the hip, which was at first thought to be a match-box dermatitis. However, the patient reported an accident in which he was struck on the other side and fell on the side which presents the lesion. At presentation the skin itself seemed to show decided infiltration. At times it was red and cyanosed, and faded gradually with the surrounding tissues.

DISCUSSION

DR. MICHELSON: One condition comes to my mind, the erysipeloid of Rosenbach.

DR. OLSON: Trimble has recently described the syndrome erythema elevatum diutinum which corresponds to the lesion presented in this patient. Erythema elevatum diutinum consists of a large plaque eruption thickest in the center, with the infiltration shading off at the border. It occurs especially in elderly men. Microscopically a simple fibrosis can be seen, differentiating it from many other similar lesions.

DR. COOK: I did not notice any elevation of the skin in this case.

D. D. TURNACLIFF, M.D., Secretary.

Book Review

MODERN CLINICAL SYPHILOLOGY. By JOHN H. STOKES, M.D., Professor of Dermatology and Syphilology in the School of Medicine, University of Pennsylvania, with the cooperation of PAUL A. O'LEARY, M.D., and W. H. GOECKERMANN, M.D., Section of Dermatology and Syphilology, The Mayo Clinic, and LOREN W. SHAFER, M.D., and CLEVELAND J. WHITE, M.D., Department of Dermatology and Syphilology, School of Medicine, University of Pennsylvania. With 865 illustrations and text figures. Price, $12 net. Philadelphia and London: W. B. Saunders Company, 1926.

Stokes' work on syphilis may be said to justify the high anticipations of those who have known it was in course of preparation. It is a highly valuable contribution to the subject and has certain features which give it the stamp of originality in both form and substance. No one else has used the same approach to the subject of syphilis, and we know of no other approach which equals Stokes', in the manner in which it, first, gives a broad orientation of the subject and, second, fills in the details so that their bearing on the problems in hand is brought out.

The plan of the work may be suggested by an analysis of the table of contents: The first five chapters are devoted to a broad consideration of the general facts of syphilis from pathology to treatment; that is, to the principles of syphilology. After this orientation of the general subject the work is devoted to developing the details of the knowledge of it. In the next six chapters the various subjects included in the treatment of syphilis in general are considered in extenso. The next three chapters take up the diagnosis and treatment of primary and secondary syphilis. These are followed by seven chapters on late syphilis. In these seven chapters are considered all types of late syphilis, from syphilis of the skin and bones, through visceral and vascular syphilis, to syphilis of the nervous system and congenital syphilis. The last two chapters are devoted respectively to miscellaneous aspects and case memorandums, and to the public and social aspects of syphilis.

The author has thus laid a broad plan and then filled in the details with minute care. To every topic that he takes up he gives the most thorough and fullest consideration. For example, in the preliminary chapters on the principle of syphilis, an entire chapter is devoted to the physical examination for syphilis. Throughout the book this meticulous attention to details is in evidence. The book is a mine of facts concerning syphilis.

But this attention to detail is only one of the striking features of the book. Another is the arrangement of facts to show their rational relation to other facts. As an illustration of this one may refer to his comparative summary of the clinical course and pathologic background of syphilis on pages 22 and 23. Here are given in parallel columns the clinical facts of the different phases of syphilis and the pathologic backgrounds which account for them. We know of no equally illuminating way of giving the reader a grasp of the facts of syphilis and the reasons for them. There are a great many detail tables of this sort throughout the work. They are a feature of high value. They emphasize the chief points, crystalize the reader's knowledge and give him a rational background for his knowledge.

The work shows that the author is a master of his subject. It is a feat to have a complete grasp of the present knowledge of syphilis. One who did not have this could not have presented the facts, and one without a philosophic mind could not have ordered the presentation or have developed it, as Stokes has done.

The book is invaluable. Surely it will be read by all serious students of syphilis and be an influence for permanent good in this enormously important field.

A book like this means that the writer has put his heart's blood into it. Stokes has the spirit of a crusader and he has shown that spirit in his study of syphilis. This work justifies all his zeal and effort in preparing it, and the immense amount of labor and thought that he has given to the subject of syphilis in all its phases. It should be a recompense to him for these hard endeavors.

Index to Current Literature

DERMATOLOGY

Acute Disseminated Lupus Erythematodes in a Woman. A. Berglund, Hygeia **88**:545 (July 31) 1926.
Acute Polymyositis or Dermatomyositis. A. Sega, Arch. di pat. e clin. med. **5**:213 (Aug.) 1926.
Allergic Test in Leprosy. W. H. Hoffmann, München. med. Wchnschr. **73**:1269 (July 30) 1926.
Atrophic Dermatitis. A. Versari, Riforma med. **42**:651 (July 12) 1926.
Atrophy of Pituitary in Skin Atrophy. L. Wertheim, Arch. f. Dermat. u. Syph. **150**:464 (July 1) 1926.

"Biology of Skin." Vollmer, Klin. Wchnschr. **5**:1371 (July 23) 1926.
Bismuth in Treatment of Yaws. D. Mendoza, Rev. de med. y. cirug. **9**:92 (May) 1926.
Bullous Ichithyosiform Erythroderma: Two Cases. W. H. Goeckerman, Arch. Dermat. & Syph. **14**:158 (Aug.) 1926.

Carcinoma of the Skin. A. Ravogli, Dermat. Wchnschr. **83**:1026 (July 10) 1926.
Chemical Composition of Human Epidermis. Y. Jono, J. Oriental Med. **5**:12 (July) 1926.
Climatic Dermatitis. E. Escomel, Gac. méd. de Carácas **33**:131 (May 15) 1926.
Coffee, Alcohol and Scurvy. F. V. v. Hahn, Deutsche med. Wchnschr. **52**:1300 (July 30) 1926.
Creeping Eruption: Report of First Case from Manitoba. K. J. Austmann, J. A. M. A. **87**:1196 (Oct. 9) 1926.
Cutaneous Manifestations of Protein Allergy. G. Piness and H. Miller, Arch. Dermat. & Syph. **14**:145 (Aug.) 1926.

Degkwitz' Measles Serum. A. Reuter, Med. Klin. **22**:1181 (July 30) 1926.
Dermatology in Children in 1925. M. Pillon, Rev. franç. de Pédiat. **2**:360 (July) 1926.
Dermatosis from Dyed Fur. G. Thibierge and J. Lacassagne, Presse méd. **34**:1009 (Aug. 11) 1926.
Dermatosis and Ascaridiasis. A. Cederberg, Arch. f. Dermat. u. Syph. **150**:393 (July 1) 1926.
Diagnosis and Therapy of Swine Erysipelas Arthritis. F. Barth, Beitr. z. klin. Chir. **137**:170, 1926.
Dick Test and Immunization. B. Johan, Deutsche med. Wchnschr. **52**:1296 (July 30) 1926.

Eczema. A. Whitfield, Brit. M. J. **2**:332 (Aug. 21) 1926.
Efficacy of Vaccination Against Smallpox: Recent Experience. C. L. Scamman and O. A. Dudley, Boston M. & S. J. **195**:538 (Sept. 9) 1926.
Erythema Nodosum and Tuberculosis. Kadri Rachid, Presse méd. **34**:1095 (Aug. 28) 1926.
Erythema Nodosum and Tuberculosis. Bydal, Norsk. Mag. f. Lægevidensk. **87**:675 (Aug.) 1926.
Etiologic Unity or Duality of Herpes Zoster and Chickenpox. O. Cozzolino, Pediatria **34**:809 (Aug. 1) 1926.
Etiology of Pellagra. J. Goldberger, Medicine **5**:79 (May) 1926.
Experimental Human Herpes. P. Teissier et al., J. physiol. et de path. gén. **24**:271; 301; 316 (April) 1926.

Febrile Types of Erythema Multiforme and Erythema Nodosum. G. Blumer, Boston M. & S. J. **195**:515 (Sept. 9) 1926.
Functional Tests in Eczema. Evening, Dermat. Wchnschr. **83**:1355 (Sept. 11) 1926.

Genital Furuncle Turns Scale in Favor of Cesarean Section. G. Kubig, Zentralbl. f. Gynak. **50**:2022 (July 31) 1926.

Golden Anniversary of the American Dermatological Association. Current Comment, J. A. M. A. **87**:1397 (Oct. 23) 1926.
Gold Treatment of Lupus Erythematodes. S. Rothman, Beitr. z. Klin. d. Tuberk. **63**:906 (July 21) 1926.
Gold Treatment of Skin Tuberculosis. G. Hufschmitt, Paris méd. **61**:135 (Aug. 14) 1926.

Herpetic Viruses and Encephalitis. M. Flexner and J. A. Flexner, Kentucky M. J. **24**:375 (Aug.) 1926.
Hyperglycemia with Skin Diseases. L. Hudelo and R. Kourilsky, Presse méd. **34**:1041 (Aug. 18) 1926.
Hypersensitiveness and Hyposensitiveness. R. Doerr, Arch. f. Dermat. u. Syph. **150**:509 (July 1) 1926.

Inefficiency of Most of the Commonly Used Skin Antiseptics. Martin B. Tinker and Henry B. Sutton, J. A. M. A. **87**:1347 (Oct. 23) 1926.
Infantile Kala Azar in Syria. J. K. Hitti, J. A. M. A. **87**:1302 (Oct. 16) 1926.
Infection with Aspergillus Versicolor. A. E. Steele, Boston M. & S. J. **195**:536 (Sept. 9) 1926.
Influence of Periarterial Sympathectomy on Skin Grafts. J. Dalger, Lyon chir. **23**:451 (July-Aug.) 1926.
Intracutaneous Salt Solution Wheal Test. W. G. Stern and M. B. Cohen, J. A. M. A. **87**:1355 (Oct. 23) 1926.
Intradermal Vaccination. M. Bussel and R. Stankiewicz, Rev. franç. de Pédiat. **2**:293 (July) 1926.
Iodine Treatment of Scarlet Fever. H. J. Novack, M. J. & Record **124**:267 (Sept. 1) 1926.
Itching and Eczema. S. Rothman, Arch. f. Dermat. u. Syph. **150**:489 (July 1) 1926.

Kahn Precipitation Reaction in Leprosy: III. E. V. Pineda and E. Roxas-Pineda, Philippine J. Sc. **30**:295 (July) 1926.
Kahn Test in Leprosy. M. V. Argüelles, Philippine J. Sc. **30**:275 (July) 1926.

Leprosy in Korea. R. M. Wilson, J. A. M. A. **87**:1211 (Oct. 9) 1926.
Lupus Carcinoma. F. Dietel, Beitr. z. klin. d. Tuberk. **63**:667 (July 21) 1926.
Lymphogranulomatosis. F. A. Risquez, Gac. méd. de Carácas **33**:145 (May 31) 1926.

Measles. F. Cirajas Labajo, Arch. españ. de pediat. **10**:465 (Aug.) 1926.
Measles Prophylaxis: Report on the Use of Immune Goat Serum. Archibald L. Hoyne and B. M. Gasul, J. A. M. A. **87**:1185 (Oct. 9) 1926.
Measles Serum. B. de Rudder, Klin. Wchnschr. **5**:1406 (July 30) 1926.
Measles Serum from Animals. R. Degkwitz, Klin. Wchnschr. **5**:1361 (July 23) 1926.
Measles Serum from Animals. W. Schmidt, Deutsche med. Wchnschr. **52**:1303 (July 30) 1926.
Mode of Infection in Coccidioidal Granuloma. F. E. Ahlfeldt, Arch. Path. **2**:206 (Aug.) 1926.
Modern Methods of Treating American Leishmaniasis of the Skin. G. Patterson, Prensa méd. argent. **13**:177 (July 20) 1926.
Mycoses of Skin. F. Pinkus, Med. Klin. **22**:1232 (Aug 6) 1926.

Neuropathic Type of Eczema. E. Brill, Arch. f. Dermat. u. Syph. **150**:529 (July 1) 1926.

Pellagra in Cuba. V. Pardo Castelló and J. J. Mestre, Rec. méd. cubana. **37**:703 (July) 1926.
Problem of Measles: Possible Solution. R. H. Cowley, Kentucky M. J. **24**:365 (Aug.) 1926.
Prognosis of Furuncles. J. Schütz, München. med. Wchnschr. **73**:1235 (July 23) 1926.
Prophylaxis of Measles. J. A. Toomey, Am. J. Dis. Child. **32**:401 (Sept.) 1926.

Rash in Scarlet Fever Delayed Till Fifth Day. L. P. Philips, Brit. M. J. **2**:343 (Aug. 21) 1926.

Recent Research on Measles. G. Caronia, Arch. españ. de pediat. **10**:449 (Aug.) 1926.
Regulation of Blood Flow Through Capillaries of Human Skin. T. Lewis, Heart **13**:1 (Aug.) 1926.
Relative Value of Tuberculin Skin Tests. F. F. Tisdall and A. Brown, Canad. M. A. J. **16**:939 (Aug.) 1926.
Results of Use of Scarlet Fever Antitoxin. H. B. Cushing, Canad. M. A. J. **16**:936 (Aug.) 1926.

Sacral Abscesses and Dermoid Formations. F. Oehlecker, Deutsche Ztschr. f. Chir. **197**:280 (July) 1926.
Sanocrysin Eruption. C. Mainini and A. Alvarez, Semana méd. **2**:204 (July 22) 1926.
Scarlet Fever. M. S. Lewis, J. Tennessee M. A. **19**:5 (May) 1926.
Scarlet Fever Antitoxin in Case of Endocarditis and Myocarditis. E. S. Toogood, Lancet **2**:545 (Sept. 11) 1926.
Scarlet Fever Complicated by Peritonitis. Ruth E. Taylor, J. A. M. A. **87**:1559 (Nov. 6) 1926.
Scarlet Fever Control. W. H. Enneis, Southwestern Med. **10**:347 (Aug.) 1926.
Scurvy in an Infant. Rámon Garin Sanchez, Arch. latino-am. de pediat. **20**:326 (June) 1926.
Serology of Leprosy. A. Raevskiy and J. Braul, Vestnik Microbiol. i Epidemiol. **5**:139, 1926.
Skin Changes in Pulmonary Tuberculosis. A. Jordan, Arch. f. Dermat. u. Syph. **150**:451 (July 1) 1926.
Skin in Children. J. Velasco Pajares, Pediatria españ. **15**:125 (May) 1926.
Skin Disease and Endocrine Disturbance. B. Bloch, Schweiz. med. Wchnschr. **56**:753 (Aug. 7) 1926.
Skin Pathology. S. Gordon, M. J. & Record **124**:213 (Aug. 18) 1926.
Skin Tests in Allergic Diseases. W. Storm van Leeuwen, Klin. Wchnschr. **5**:1525 (Aug. 13) 1926.
Skin Tuberculosis in Pomerania. W. Leipold, Beitr. z. Klin. d. Tuberk. **63**:857 (July 21) 1926.
Smallpox or Chickenpox? D. J. Hulshoff Pol, Nederl. Tijdschr. v. Geneesk. **2**:758 (Aug. 14) 1926.
Sodium Chloride Metabolism in Cutaneous Burns. E. C. Davidson, Arch. Surg. **13**:262 (Aug.) 1926.
Softening of Scars. H. Kurtzahn and W. v. Bülow, Deutsche Ztschr. f. Chir. **198**:43 (Aug.) 1926.
Specialists of Skin and Venereal Diseases. O. Sprinz, Klin. Wchnschr. **5**:1589 (Aug. 20) 1926.
Specific Necrosis in the Infectious Granulomas. E. R. Long, J. A. M. A. **87**:1441 (Oct. 30) 1926.
Swine Erysipelas in Man. R. Schrapf and E. Fouquet, Presse méd. **134**:1126 (Sept. 4) 1926.

Treatment of Furunculosis. S. Levy, Deutsche med. Wchnschr. **52**:1303 (July 30) 1926.
Treatment of Psoriasis. R. W. MacKenna and E. F. Skinner, Brit. M. J. **2**:338 (Aug. 21) 1926.
Treatment of Scleroderma. F. Herzog, Med. Klin. **22**:1178 (July 30) 1926.
Treatment of Skin Diseases. Veyrières and Valette, Paris méd. **61**:132 (Aug. 14) 1926.
Treatment of Skin Tuberculosis. M. Schubert, Beitr. z. Klin. d. Tuberk. **63**:939 (July 21) 1926.
Tropical Ulcer. E. P. de Bellard, Gac. méd. de Carácas **33**:164 (June 15) 1926.
Tuberculin Test in Measles and German Measles. R. Debré and K. Papp, Compt. rend. Soc. de biol. **95**:29 (June 11) 1926.
Tumors in Skin from Foreign Bodies. H. C. S. Finsen, Hospitalstid. **69**:645 (July 8) 1926.

Urticarial Bismuth Exanthema. A. Helfer, Dermat. Wchnschr. **83**:1358 (Sept. 11) 1926.

Value of the Study of Histopathology in Cutaneous Medicine. W. J. Highman, J. A. M. A. **87**:1459 (Oct. 30) 1926.

Variations of Cutaneous Toleration for Roentgen Rays. George M. MacKee and Joseph J. Eller, J. A. M. A. **87**:1533 (Nov. 6) 1926.
Vascular Reactions of Skin to Injury: III. Some Effects of Freezing, of Cooling and of Warming. T. Lewis and W. S. Love, Heart **13**:27 (Aug.) 1926.

Yellow Fever in 1923. D. Hernandez Bautista, Rep. de med. y cirug. **17**:463 (May) 1926.
Yellow Fever Retreats from the Americas. Editorial, J. A. M. A. **87**:1480 (Oct. 30) 1926.

SYPHILOLOGY

Antoni's Blood Picture in Syphilis. Stern and Strauss, Dermat. Wchnschr. **83**:1345 (Sept. 11) 1926.
Arsphenamine Jaundice. K. Bardach, München. med. Wchnschr. **73**:1237 (July 23) 1926.
Arsphenamine Therapy. H. Th. Schreus, Arch. f. Dermat. u. Syph. **150**:402 (July 1) 1926.
Artificial Syphilitic Blood Changes. F. Heimann, Arch. f. exper. Path. u. Pharmakol. **115**:64 (Aug.) 1926.

Bilateral Gummata of Sternoclavicular Articulations. G. T. Pack and J. S. Beck, Am. J. Syph. **10**:383 (July) 1926.
Biologic Diagnosis of Inherited Syphilis. Mouriz Riesgo, Med. Ibera **20**:54 (July 17) 1926.
Bismuth in Syphilis. J. Urbach, Med. Klin. **22**:1233 (Aug. 6) 1926.
Bismuth Treatment of Syphilis. J. Urbach, Med. Klin. **22**:1194 (July 30) 1926.
Bismuth and Wassermann Reaction. D. M. Sidlick, Am. J. Syph. **10**:486 (July) 1926.
Boltz Test in Cerebrospinal Fluid After Malarial Therapy in General Paralysis. G. W. T. H. Fleming, J. Ment. Sc. **72**:376 (July) 1926.
Bone Syphilis. F. H. Dillingham and L. K. McCafferty, Am. J. Syph. **10**:373 (July) 1926.

Calcium Content of Serum in Syphilis. I. Rosen and F. Krasnow, Am. J. Syph. **10**:446 (July) 1926.
Cerebral Syphilis: Convulsions: Death. N. M. Goodman, Brit. M. J. **2**:302 (Aug. 14) 1926.
Cerebrospinal Fluid in Syphilis. F. Mras, Wien. klin. Wchnschr. **39**:950 (Aug. 12) 1926.
Chancroidal Buboes. H. G. Miskjian, J. A. M. A. **87**:1436 (Oct. 30) 1926.
Chemical Investigations on Neo-Arsphenamine: Methods of Analyses. L. Freedman, Am. J. Syph. **10**:460 (July) 1926.
Civilization, Vaccination and General Paralysis. A. Ameghino, Rev. Asoc. méd. argent. **39**:221 (June) 1926.
Clinical Application of Flocculation Reaction with Dried Japanese Fish with Blood Serum and Plasma of Syphilitic Children. S. Takeda, Oriental J. Dis. Infec. **1**:81 (July) 1926.
Comparison of Fixation at 3-6 C. and 37 C. in Over 25,000 Complement Fixation Tests for Syphilis Made with Cholesterinized Antigen. R. Gilbert, V. Langworthy and A. C. Moore, Am. J. Syph. **10**:506 (July) 1926.
Congenital Syphilis of Dementia Type. M. A. Jáureguy, Arch. latino-am. de pediat. **20**:320 (June) 1926.
Congenital Syphilitic Lesions Exclusively in the Long Bones. Encinas de la Rosa, Pediatria españ. **15**:173 (June) 1926.
Congenital Syphilis in Univitelline Twins. F. Weiss, Arch. f. Kinderh. **79**:18 (Aug. 7) 1926.
Control of Communicable Diseases Prevalent in Massachusetts with Study of Mortality Due to Them During Past Seventy-Five Years. E. G. Huber, Boston M. & S. J. **195**:539 (Sept.) 1926.
Conjugal and Familial Cardiovascular Syphilis. R. A. Bullrich, Semana méd. **2**:53 (July 8) 1926.

Detoxicated Arsphenamine. E. S. Heronimus, Arch. f. Dermat. u. Syph. **150**:459 (July 1) 1926.

Diabetes Insipidus Associated with Syphilis of Hypophysis. E. B. Fink, Endocrinology **10**:317 (May-June) 1926.
Dystrophies of the Aorta and Syphilis. M. Letulle et al., Presse méd. **34**:1089 (Aug. 28) 1926.

Early Recognition of Various Types of Syphilis. A. Crance, New York State J. Med. **26**:697 (Aug. 15) 1926.
Elements Resembling Spirochetes Found in Blood in Epidemic Encephalitis. F. Wanner, Schweiz. med. Wchnschr. **56**:767 (Aug. 7) 1926.
Experimental Transformation of Granulomas. A. Pickham, Ztschr. f. Krebsforsch. **23**:496 (July 28) 1926.

First Symptoms of Neurosyphilis. C. S. Bluemel, Am. J. Syph. **10**:420 (July) 1926.

International Comparative Statistics of General Paresis. Kolb, J. Nerv. & Ment. Dis. **64**:225 (Sept.) 1926.
Interpretation of Wassermann Reaction in Terms of Hydrogen-Ion Concentration. A. Kahn, Am. J. Syph. **10**:489 (July) 1926.
Interstitial Keratitis in Congenital Syphilis: Clinical Notes on Incidence and Treatment. W. H. Guy, J. A. M. A. **87**:1551 (Nov. 6) 1926.

Late Syphilis of Joints. H. Schlesinger, Klin. Wchnschr. **5**:1474 (Aug. 6) 1926.
Latent Syphilitic Infection and Superinfection. W. Kolle and H. Schlossberger, Deutsche med. Wchnschr. **52**:1245 (July 23) 1926.
Leukocytes in Syphilis. P. Mulzer, Klin. Wchnschr. **5**:1520 (Aug. 13) 1926.

Malaria Treatment of General Paralysis. J. Dunne, J. Ment. Sc. **72**:343 (July) 1926.
Menace of the Slightly Positive Wassermann Reaction. J. H. Mitchell, J. A. M. A. **87**:1351 (Oct. 23) 1926.
Modifications of Sachs-Georgi Test Used to Determine Incidence of Syphilis in Mental Patients. H. Smith, J. Ment. Sc. **72**:340 (July) 1926.

Nonspecific Protein Therapy in General Paralysis: Preliminary Report. M. M. Kunde, G. W. Hall and F. J. Gerty, J. A. M. A. **87**:1376 (Oct. 23) 1926.
Nonspecific Stimulation Therapy; Wassermann Reaction and Syphilis Therapy with Living (Malaria) and Dead Lipoid-Albumin Compounds. J. Schumacher, Am. J. Syph. **10**:432 (July) 1926.
Nervous Syphilis. H. V. Wildman, Jr., Am. J. Syph. **10**:429 (July) 1926.
Neurosyphilis: Intraspinal Treatment. W. C. Stoner, Am. J. Syph. **10**:439 (July) 1926.

Otomastoiditis in Syphilitics. P. Truffert, Paris méd. **61**:193 (Sept. 4) 1926.
Outfit for Collection of Serum by Physicians for Dark-Field Examination in Suspected Syphilis. W. R. Stokes and C. L. Ewing, Am. J. Syph. **10**:404 (July) 1926.

Paralysis of Divergence with Chorea and with Tabes. J. E. Lebensohn, Am. J. Ophth. **9**:684 (Sept.) 1926.
Persistent Positive Wassermann Reaction. L. G. Beinhauer, Am. J. Syph. **10**:454 (July) 1926.
Phases of Neurosyphilis. A. Myerson and M. Yorshis, Am. J. Syph. **10**:410 (July) 1926.
Precipitation with Fractions of Syphilitic Serum and Arachnoid Fluid. P. L. Kendrick and R. L. Kahn, J. Infect. Dis. **39**:202 (Sept.) 1926.
Precipitation Test in Diagnosis of Syphilis. M. Garriga, Med. Ibera **20**:50 (July 17) 1926.
Present Status of Syphilis in Children. M. Péhu, Rev. franç. de Pédiat. **2**:373 (July) 1926.
Present Status of Wassermann Reaction. P. B. Newcomb, Southwestern Med. **10**:333 (Aug.) 1926.
Preventive Treatment of Syphilis. M. Joseph, Deutsche med. Wchnschr. **52**:1262 (July 23) 1926.

Radiography of Epiphyseal Cartilages in Syphilis. M. Springer, Presse méd. **34**:1159 (Sept. 11) 1926.

Rapidly Developing Syphilitic Paraplegia. M. Chung, Arch. Dermat. & Syph. **14**:111 (Aug.) 1926.
Reducing Cost of Syphilis in Industry. W. A. Sawyer and B. J. Slater, New York State J. Med. **26**:711 (Aug. 15) 1926.
Reducing Cost of Syphilis in Industry. W. A. Sawyer and B. J. Slater, J. Indust. Hyg. **8**:317 (Aug.) 1926.
Reinfection in Syphilis. H. Reiter, Klin. Wchnschr. **5**:1356 (July 23) 1926.
Results with Tryparsamide Treatment of Neurosyphilis. C. A. Neymann and D. E. Singleton, J. Nerv. & Ment. Dis. **64**:144 (Aug.) 1926.

Significance of Gain in Weight in Malaria Treatment of General Paralysis. H. A. Bunker, Jr., Arch. Neurol. & Psychiat. **16**:329 (Sept.) 1926.
Spirochetes in Baltimore Market Oysters. V. T. Dimitroff, J. Bact. **12**:135 (Aug.) 1926.
Syphilis. R. E. Jameson, Am. J. Syph. **10**:478 (July) 1926.
Syphilis and Acute Yellow Atrophy of the Liver. A. Neddermeyer, Arch. f. Dermat. u. Syph. **150**:423 (July 1) 1926.
Syphilis and Gastropathies. H. Surmont, Médecine **7**:772 (July) 1926.
Syphilis on Medical Service. H. M. Moses, Am. J. Syph. **10**:388 (July) 1926.
Syphilis of Retinal Vessels. E. v. Hippel, Arch. f. Ophth. **117**:221 (July 21) 1926.
Syphilitic Aortitis. F. Moritz, München. med. Wchnschr. **73**:1263 (July 30) 1926.
Syphilitic Cystitis, A. Avramovici, J. d'urol. **22**:26 (July) 1926.
Syphilitic Pseudoparalysis. Engel, Deutsche med. Wchnschr. **52**:1393 (Aug. 13) 1926.
Syphilitic Stricture of Small Intestine. R. Hinz, Arch. f. klin. Chir. **141**:494 (Aug. 7) 1926.

Tertiary Syphilis of Eyelid. E. S. Connell, Am. J. Ophth. **9**:608 (Aug.) 1926.
Treatment of General Paralysis by Fever-Producing Methods, Editorial, J. A. M. A. **87**:1394 (Oct. 23) 1926.
Treatment of Syphilis. F. W. Cregor and F. M. Gastineau, J. Indiana M. A. **19**:305 (Aug. 15) 1926.
Treatment of Syphilis of Nervous System. P. Bassoe, Illinois M. J. **50**:111 (Aug.) 1926.
Treatment of Tabes and Paralysis. K. Löwenstein, Deutsche med. Wchnschr. **52**:1393 (Aug. 13) 1926.
Turbidity Reactions in Serodiagnosis of Syphilis. S. Mutermilch, Presse méd. **34**:1108 (Sept. 1) 1926.

Wassermann Reaction After Bile Injections. D. Kirschenblatt and A. Narinjan, Addendum. F. Klopstock, Deutsche med. Wchnschr. **52**:1298 (July 30) 1926.
Wassermann Test: XXI. Production of Syphilitic Reagin. D. L. Belding, Am. J. Syph. **10**:500 (July) 1926.
Whittingham (W.) Strain of Artificially Induced Malaria in Treatment of General Paralysis and Tabes Dorsalis. A. R. Grant and J. D. Silverston, J. Ment. Sc. **72**:346 (July) 1926.
Whooping Cough in Congenital Syphilis. W. Mikulowski, Rev. franç. de Pédiat. **2**:332 (July) 1926.

INDEX TO VOLUME 14

The star (*) preceding the page number indicates an original article in THE ARCHIVES. No author entries are made except for original articles. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

PAGE

Acanthosis nigricans....92, 618, 619
nigricans and cancer.... 739
Acid-alkali equilibrium and eczema.... 456
Acidosis, eczema, insulin, and.... 582
Acne agminata.... 194
oil, in lathe workers.... 459
varioliformis 207
vulgaris, infantile *12
vulgaris, with keloid formation.... 497
Aconitine, treatment of circumscribed pruritus (neurodermite) by ionization with 453
Acrodermatitis chronica atrophicans: See Atrophia
perstans, of Hallopeau....93, 612
Acrodynia 738
in adults.... 727
vaccine therapy of.... 736
Acromegaly, cutis verticis gyrata.... 63
Actinomycosis in Mexico.... 326
Adenocarcinoma 593
Adenoma sebaceum....466, 483, 591
mucous adenomas.... 494
Albinism: See Leukoderma
Aleppo Boil: See Leishmaniasis
Allergy: See also Anaphylaxis
cutaneous manifestations of protein allergy *145
Alopecia 745
generalized 192
permanent, following roentgen-ray treatment 339
syphilitic 89
American Dermatological Association, fiftieth anniversary meeting of.... 316
founding of *371
Amyloidosis, localized, of skin with osseous metaplasia 460
Anaphylaxis, primary allergy and shock... 329
to milk.... 329
Anemia following injections of neoarsphenamine 455
Gaucher's disease with cutaneous lesions. 322
pernicious anemia—glossitis.... 222
Angina, iodide eruption; Vincent's angina. 621
Angiokeratoma (Mibelli) 497
Angiolupoid, pathogenesis of.... 327
Angioma: See also Nevus
and verrucous nevus.... 230
nevus vasculosus 475
serpiginosum 363
Anthrax, edematous, of face resulting in meningitis 446
treatment 201
Aorta, syphilis of aorta and heart.... 577
syphilitic aortitis.... 202
Arachnidism: See Spider poisoning
Argyria from argyrol.... 484
pigmentation of lips and buccal mucosa; argyria? 493

PAGE

Arsenic, pigmentation and keratoderma from 451
pigmentation from.... 612
rheumatic pains in course of treatment with arsenic and bismuth combined... 581
Arsphenamine: See also Neoarsphenamine
accidents 574
controlling the odor and taste of.... 63
dermatitis 82, 223, 334
dermatitis, fixed *713
dermatitis, sodium thiosulphate in.... 65
hepatitis from; maculopapular syphilid.. 474
reactions, alleged stimulation of autonomic nervous system in....*382
treatment, control of, by liver function tests 587
Arthritis, ioderma; acute arthritis (syphilitic) 77
Asthma and ichthyosis.... 456
Atrophia, acrodermatitis chronica atrophicans 74, 459
acrodermatitis chronica atrophicans, with neuritis 332
cutaneous 471
macular 740
macular, idiopathic.... 626
reticular 94
Ayres, S., Jr.: Infantile acne vulgaris; report of 3 cases.... *12

Bazin's Disease: See Erythema induratum
Becker, S. W.: Generalized telangiectasia; clinical study; etiology and pathology.*387
Beeson, B. B.: Granulosis rubra nasi (Jadassohn) *256
Treatment of syphilis according to Jacques de Bethencourt, 1527; extracts from French translation by Alfred Fournier, 1871....*427
Beinhauer, L. G.: Trichotillomania.... *59
de Bethencourt, Jacques, treatment of syphilis according to; extracts from French translation by Alfred Fournier, 1871.*427
Bismuth abscess.... 197
carbonate, arterial embolism due to intramuscular injection of.... 461
death due to.... 737
hydroxide, analysis of organic deposit obtained by opening abscess formed after intramuscular injection of.... 452
hydroxide, ecchymotic and vesicular plaque following an intragluteal injection of curalues.... 454
hydroxide, local accidents following injections of 197
rheumatic pains in course of treatment with arsenic and bismuth combined... 581
toxicology of bismuth compound of series of bismuthates 451
Blastomycosis 194, 470
cutaneous 584
pathologic anatomy of Gilchrist's blastomycosis of skin.... 462
Blood, chemistry of, in some dermatoses.. 66
influence of chemical light baths on bactericidal processes in.... 463
sugar in dermatoses....201, 581, 581
Bodily structure and skin diseases.... 327
Bone resorption, extensive, calcification of skin, including epiderm, in connection with *503

BOOK REVIEWS: PAGE

Diseases of the Skin; L. Sutton..... 104
Eczema and Urticaria; Pulay..... 237
Faculté de Médecine de Strasbourg Travaux de la Clinique des Maladies Cutanées et Syphilitiques; L. M. Pautrier 364
Förhandlingar vid den Nordiska Dermatologiska Föreningens Möte, Helsingfors, 26-28 Augusti, 1924..... 364
Fundamentals of Dermatology; A. Schalek 627
Jahresbericht über Haut-und Geschlechts-Krankheiten sowie deren Grenzgebiete, Vol. IV; Edited by O. Sprinz..... 627
Modern Clinical Syphilology; J. H. Stokes 747
National Medical Manual; Year Book of Treatment and Practitioners' Index... 104
Bowen's Disease.....194, 585
Breast, double syphilitic mastitis in course of multiple visceral syphilis..... 323
Bromoderma, intravenous administration of sodium chloride 578
Brown, H.: Urobilinogen determination in treated syphilitic patients.....*434
Bubo, atypical chancroidal bubos..... 459
bacteriotherapy of chancroidal bubo; treatment by typhoid-paratyphoid vaccine 454

Calcification of skin, including epiderm, in connection with extensive bone resorption *503
Camphorated oil infiltrates..... 465
Cancer: See also Epithelioma
acanthosis nigricans and..... 739
adenocarcinoma 593
biologic action of hard and soft roentgen rays in relation to skin cancer..... 730
facial nevocarcinoma, recurring after excision 582
generalized carcinomatosis 453
itching as initial symptom of mammary cancer 731
metastasis, mechanism of..... 198
mule spinners' cancer, cause of..... 442
multiple occupational squamous carcinoma in gunsmith..... 580
of floor of mouth..... 85
of lip..... 588
of lip, and syphilitic glossitis..... 495
prickle cell carcinoma in boy..... 93
radium applicator for treatment of..... 193
scar carcinoma 197
treatment, colloidal lead in..... 579
treatment of external cancer..... 589
vitamin imbalance, roentgen-ray activity and 447
Cerebrospinal Fluid, human, in system and diffuse degenerative disease involving nervous system..... 319
refractometric and viscosimetric indexes of 319
tests, analysis of..... 578
Chancre, anal chancre?..... 221
double, of cheek and right eyebrow..... 459
initial lesion of pubic region..... 224
multiple 236
of base of thumb..... 197
of eyebrow (right side), with secondary lesions 89
of prolonged incubation..... 460
of vagina..... 66
sycosiform, of chin and mucous surface of lower lip..... 197
unusual location of primary lesions..... 583
Chancroid, atypical chancroidal bubos..... 459
bacteriotherapy of chancroidal bubo; treatment by typhoid-paratyphoid vaccine.. 454
microscopic search for Ducrey bacillus.. 582
of eyelid..... 446
of tongue..... 460
serpiginous 75

PAGE

Chickenpox and zoster occurring simultaneously 455
nuclear inclusions in testicles of monkeys injected with tissue of human varicella lesions 321
two new cases establishing relationship of origin of zoster and..... 583
Chigger fleas, entomologic episode of East African campaign 199
Chilblains, erythema pernio..... 497
ulcerative pernio, galvanization of thyroid gland in..... 460
Children, skin in..... 738
Cholesterin in diseases of skin..... 450
Chung, Mon-Fah: Study of 34 cases of rapidly developing syphilitic paraplegia *111
Climatic dermatitis..... 738
Cole, H. N.: Excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683
Colloid degeneration of skin..... 742
Combes, F. C., Jr.: Lymphosarcoma; report of 2 cases..... *28
Comedo formation with general endocrinopathy 620
Condyloma acuminatum 213
Cooke, J. V.: Dermatitis of diaper region in infants (Jacquet dermatitis).....*539
Coolidge, W. D., medal awarded to..... 572
Copper sulphate, alcoholic solution of, in dermatology 581
sulphate, liquor of Alibour in pityriasis capitis 581
Creeping eruption in Manitoba..... 727
Curalues: See under Bismuth
Cutis verticis gyrata.....63, 624
Cyanosis, erythema induratum and acrocyanosis 325
failure of tuberculin therapy in cyanotic puffiness of extremities..... 460
Cyst, retention, of lower lip..... 742

Dandruff, skin tests with..... 590
study of dandruff and pityrosporon of Malassez *270
Dermatitis, climatic dermatitis..... 738
due to heat sensitization, and dermatitis dysmenorrheica 492
dysmenorrheica 492
epithelioma of ear; previously reported as chondrodermatitis chronica helicis..... 103
exfoliativa 206, 475, 595
factitious 359
herpetiformis 339, 472, 621
herpetiformis, of Duhring-Brocq of cicatricial evolution..... 449
infectious eczematoid 351
interdigitalis cryptococcica 585
lichenoid and purpuric dermatitis, unnamed 457
light dermatitis and pigmentation, inhibition by procaine..... 730
medicamentosa: See Drug eruptions and under names of drugs as Arsphenamine; Iodine; Phenolphthalein, etc.
newspaper ink dermatitis..... 472
occupational, from wood turpentine..... 319
of diaper region in infants (Jacquet dermatitis) *539
purpuric and pigmented lichenoid dermatitis 196
radiodermatitis, chronic 474
recurrent erysipelas of legs with dermatitis of feet *679
scrofulosa 194
tear gas dermatitis 95
vegetans and pyoderma..... 212

PAGE

Dermatology, first chair in, at Harvard University 723
graduate courses in medical subjects and residencies in specialties..... 572
index to current literature.....105, 239, 365, 498, 628, 749

Dermatomycosis: see Mycosis

Dermatomyositis 729

Dermatoses, abnormal secretions of cutaneous glands and pathologic changes in skin 463
blood chemistry in..... 66
blood sugar in 201, 581
blood sugar relations with..... 581
bodily structure and..... 327
cholesteremia and glycemia in..... 729
cholesterin in 450
localization of hematogenous skin infections 739
progressive pigmentary, Schamberg's.....*53, 225, 497, 625

Dermographism 452

Dermophytes, demonstration of cultures and photographs 214

Desquamation, seasonal, of hands and feet. 589

Diabetes Mellitus, psoriasis and diabetic heredity 454
scleroderma with, controlled by insulin.. 613
syphilitic 66

Diaper region, dermatitis of (Jacquet dermatitis) *539

Diathermy, endothermy and combined therapy in dermatology..... 448

Dresel, I.: Unusual eruption due to organism of Monilia group; preliminary report *280

Drug eruptions, skin manifestations seen in general hospital *693

Ducrey bacillus: See Chancroid

Dyes, hair dyes and their toxic effects; classification and description.....*136

Dyshidroses, salicylic acid and certain vegetables, called depuratives, as provocatives of 451

Dysinsulinism, hypo-adrenalism and, of acquired or congenitally syphilitic origin; new pluriglandular syndrome.... 324

Dysmenorrhea dermatitis and dermatitis due to heat sensitization..... 492
dermatitis dysmenorrheica..... 492

Eczema, acid-alkali equilibrium and..... 456
acidosis, insulin and..... 582
behavior of injected uric acid in patients with *308
focal infections in etiology of..... 319
infectious eczematoid dermatitis-infantile eczema 88
relation of end-products of protein metabolism to *294
sympatheticotonia; eczema..... 610

Edema, angioneurotic 64
angioneurotic, of penis and scrotum.... 579
angioneurotic, psychology of..... 202
sclerous, edematous erythema with myopathy and myasthenia..... 324

Elephantiasis, sporadic 492

Embolism, arterial, due to intramuscular injection of bismuth carbonate..... 461

Encephalitis, immunological studies with herpes virus with..... 580
varicella, and vaccinia encephalitis..... 736

Endocrines, hypofunction, folliculitis associated with..... 481
comedo formation associated with general endocrinopathy 620
nails and endocrines..... 329

Epidermolysis bullosa in sisters..... 591

PAGE

Epithelioma 333, 335, 611, 743
case for diagnosis; epithelioma of chancre of lip 353
diffuse erythrokeratoderma; secondary epitheliomatosis 461
multiple benign cystic..... 620
of ear; previously reported as chondrodermatitis chronica helicis..... 103
of floor of mouth..... 223
of mouth 744
prickle cell, of face, in girl..... 458
rare epitheliomas of skin..... 67
syphilitic interstitial glossitis, leukoplakia and 235

Epstein, N. N.: Rose bengal test for liver function; with particular reference to its use in therapy of syphilis.....*122

Erysipelas; etiology, and treatment with erysipelas antistreptococcic serum..... 192
lupus vulgaris with extensive exacerbation following 462
recurrent, of legs with dermatitis of feet *679

Erysipeloid, distinctive and severe form of, among fish handlers, demonstration of bacillus of swine erysipelas.....*662

Erythema ab igne.....224, 478
annulare 215
annulare, centrifugal..... 582
case for diagnosis; elevatum diutinum. 622
case for diagnosis; erythema multiforme, or syphilis? 340
case for diagnosis; erythematobullous eruption 76
case for diagnosis; toxic erythema?.... 348
case for diagnosis; toxic erythema? pityriasis rosea?..... 344
induratum.....468, 496, 624, 745
induratum, and acrocyanosis..... 325
induratum; Bazin's disease..... 336
induratum, livedo reticularis and..... 72
induratum, of Bazin, type of sarcoid approaching 461
induratum scrofulosorum; Bazin's disease 82
infiltrative, or erythematocyanotic infiltrations of neuro-endocrine cases..... 460
multiforme 359, 473
multiforme, bullous 337
multiforme, chronic 593
multiforme, complicating vaccination.... 64
multiforme, followed by leukemia..... 324
multiforme, lupus erythematosus..... 464
multiforme perstans 617
nodosum 234
nodosum, and tuberculosis..... 200
pernio 497
sclerous, edematous, with myopathy and myasthenia 324
telangiectodes, xanthoma with parapsoriasis and 622

Erythroderma, bullous ichthyosiform; 2 cases *158
in plaques resembling lichen planus, with atrophic and sclerodermatous tendency. 455

Erythromelalgia; thermalgia 612

Esophylaxis: See under Immunity

Excoriations, neurotic; case for diagnosis.. 352

"Face Peel," fatal..... 723

Farmer's skin and ichthyosis..... 477

Favus 493
after roentgen-ray treatment..... 338
due to achorion gypseum..... 66

Fibrosarcoma, cutaneous 325, 452

Fish handlers, distinctive and severe form of erysipeloid among; demonstration of bacillus of swine erysipelas.....*662

Folliculitis, associated with endocrine hypofunction 481
decalvans 331, 494
decalvans, treated with roentgen rays... 338

PAGE

Fordyce's Disease of penis..... *50
Fox, G. H.: Founding of the American Dermatological Association.....*371
Fox-Fordyce syndrome.....*46, 591, 618
Frambesia and syphilis, immunologic relations between 732
frambesiform syphilid 602
serodiagnosis of syphilis and..... 732
yaws 345
Fungus: See also Blastomycosis; Mycosis, etc.
biochemical reactions of..... 580

Gammel, J. A.: Excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683
Gangrene of scalp from bleaching hair.... 201
Gas, tear gas dermatitis..... 95
Gaucher's Disease: See Anemia
Glossitis; pernicious anemia..... 222
syphilitic, and carcinoma of lip..... 495
syphilitic interstitial; leukoplakia and epithelioma 235
Goeckerman, W. H.: Bullous ichthyosiform erythroderma; report of 2 cases.....*158
Fordyce's condition of penis; report of case *50
Treatment of neurosyphilis by malaria..*550
Goiter, exophthalmic, syphilitic, frequency and treatment of..... 323
Goldenberg, H.: Skin manifestations seen in a general hospital.....*693
Goldzieher, M.: Granuloma inguinale; clinical observations, vaccine treatment and allergic reactions *14
Gonorrhea, acute, subacute syphilitic phlebitis in 452
Goodman, H.: Fox-Fordyce syndrome..... *46
Gout versus xanthoma..... 737
Graduate courses in medical subjects and residencies in specialties..... 572
Granuloma annulare 89
annulare? case for diagnosis..... 216
annulare, of hand..... 457
coccidiodes 596
fungoides: See Mycosis fungoides
giant mycotic, of scalp..... 585
inguinale: See also Lymphogranulomatosis inguinalis
inguinale 101, 198, 223
inguinale; clinical observations, vaccine treatment and allergic reactions..... *14
inguinale; Leishman-Donovan bodies demonstrated 336
inguinale, with lesions of face and intraoral lesions 613
pediculatum, experimental 326
pyogenic 742
pyogenicum (correction) 565
tricophyticum (Majocchi) 584
Granulosis rubra nasi (Jadassohn); report of case *256
Gray, A. M. H.: Sclerema neonatorum..*635
Greenbaum, S. S.: Urobilinogen determination in treated syphilitic patients....*434
Gummas, multiple..... 89
multiple, with negative Wassermann reaction 478
of tongue with possible malignancy..... 96
ulcerating, of dorsal surface of tongue simulating carcinoma..... 87

Hair, bleaching, gangrene of scalp from... 201
dyes, and their toxic effects; classification and description.....*136
dyes, injury from..... 442
removal, recipe for epilating wax..... 579
removal, suit follows "permanent shave". 571
removal, with thallium.....463, 584

PAGE

Hanzlik, P. J.: Alleged stimulation of autonomic nervous system in arsphenamine reactions.....*382
Harkins, M. J.: Distinctive and severe form of erysipeloid among fish handlers; clinical and laboratory studies; demonstration of bacillus of swine erysipelas *662
Hazen, H. H.: Fixed arsphenamine dermatitis *713
Heat and x-ray exposures, alternating, skin reactions to 320
Hemangioma and lymphangioma..... 85
Hemorrhage in von Recklinghausen's disease 448
Herpes, experimental human..... 728
recurrent 736
recurrent, of face in girl, with positive inoculation of rabbit's cornea..... 459
simplex, is aphthous stomatitis due to virus of?.....*439
virus, immunological studies of, with consideration of herpes-encephalitis problem 580
vulvae, and ulcus vulvae acutum..... 722
zoster, and varicella.....455, 583
zoster, etiology of..... 320
zoster, intercostal, transient keratosis pilaris following..... 323
zoster, of bladder..... 736
Hospitals providing approved residencies in certain specialties..... 572
Hydroa aestivale.....210, 225, 342, 615, 615

Ichthyosis and asthma..... 456
and "farmers' skin"..... 477
bullous ichthyosiform erythroderma.....*158
case for diagnosis..... 479
hystrix 743
Immunity, significance of cutaneous stimuli for protection of body against infection *4
Impetigo (?), case for diagnosis..... 73
Infection, focal, rôle in etiology of eczema. 319
Insulin, cholesteremia and glycemia in dermatoses 729
cholesterinemia and glycemia in xanthoma; insulin therapy..... 458
eczema, acidosis and..... 582
in psoriasis 454, 454, 456
pruritus in diabetic patient treated with intramuscular injections of..... 490
Iodin, case for diagnosis: iododerma or miliary tuberculosis..... 357
changes in skin induced by..... 729
colloidal suspensions of..... 455
dermatitis medicamentosa..... 336
eruption; case for diagnosis..... 480
eruption, pompholyx, from external application 322
eruption; Vincent's angina..... 621
ioderma; acute arthritis (syphilitic)..... 77
iododerma, bullous and vegetating, fatal case of 453
Ionization, electric, treatment of pruritus ani by *560

Jaundice, arsphenamine; maculopapular syphilid 474
syphilitic, in primary period..... 583

Kahn Test, daily variations in complement fixation (Kolmer) and precipitation (Kahn) reactions in the untreated syphilitic 578
error of basing diagnosis on, alone..... 64
microscopic slide precipitation test for syphilis 63
Kala-Azar: See Leishmaniasis
Keloids 93
scar carcinoma 197

PAGE

Keratitis, interstitial 577

Keratoderma 206
case for diagnosis..... 226
diffuse erythrokeratoderma; secondary epitheliomatosis 461
disseminated palmar and plantar.....*655
of arsenical origin..... 451
verrucous nodular 453

Keratosis pilaris 468
follicularies; Darier's disease..... 356
palmaris et plantaris..... 618
pilaris, transient, following intercostal zoster 323

Kidney tumor with congenital syphilis.... 738

Klauder, J. V.: Distinctive and severe form of erysipeloid among fish handlers; clinical and laboratory studies; demonstration of bacillus of swine erysipelas *662

Lead, colloidal, in treatment of cancer.... 579

Lehman, E. P.: Recklinghausen's neurofibromatosis and skeleton; plea for complete study of disease.....*178

Leiomyoma, multiple, of skin..... 65

Leishmaniasis; Aleppo boil..... 361
cutaneous 91
reports of Kala-Azar commission, 1924-1925 195

Leprosy 466, 489
cure of 590
diagnosis and symptomatology of; pigmented neuroleprids 68
in 14 months old child..... 580
in Korea..... 727
is curable..... 67
maculo-anesthetic 90
nodular 90, 90
origin of lepra cell..... 320
specific skin reaction in..... 731
treatment with oil of carpotroche braziliensis 461
Wassermann and Sachs-Georgi reactions in 67

Leukemia, acute lymphatic..... 598
erythema multiforme followed by..... 324
ulcerating leukemic tumor of forearm.. 197

Leukoderma: See also Vitiligo
congenital partial albinism *173

Leukoplakia, syphilitic interstitial glossitis and epithelioma 235

Lichen and lichenoid dermatitis..... 583
nitidus 321, 485
planus 743
planus, and lichen spinulosus..... 350
planus atrophicus 222
planus atrophicus, macular atrophy of.. 80
planus, case for diagnosis: lichen planus? pityriasis rosea? syphilis?..... 348
planus, case for diagnosis, pityriasis rubra pilaris? linear lichen planus?..... 476
planus (circinate type)..... 209
planus; failure of roentgenotherapy..... 457
planus, frequency of, at clinic of Strasbourg 459
planus, hypertrophic, or verrucous nevus 354
planus hypertrophicus; case for diagnosis 610
planus, in child?..... 484
planus, limited to buccal mucosa..... 624
planus, of mouth only..... 350
planus scerlosus et atrophicus..... 354
planus, zoniform 458
scrofulosorum 471
scrofulosorum (?) or secondary follicular syphilis or latent syphilis..... 601
simplex 209
spinulosus 743

Lieberthal, D.: Disseminated palmar and plantar keratoderma; report of case....*655

PAGE

Light baths, chemical, influence of, on bactericidal processes in blood..... 463

Lipomas, multiple telangiectatic, simulating von Recklinghausen's disease..... 452
symmetrical lipomatosis of extremities. 453

Liquid Air as caustic..... 581
in dermatologic therapeutics..... 581

Livedo reticularis and erythema induratum 72

Liver function, rose bengal test for; its use in therapy of syphilis.....*122
function tests, control of arsphenamine treatment by 587
urobilinogen determination in treated syphilitic patients *434

Lupus, destructive, of face and syphiloid ulcerations of arm; arsenical medication 456
erythematosus .....
.....228, 464, 469, 602, 602, 603, 603
erythematosus diffuse scleroderma..... 465
erythematosus, discoid, of back..... 484
erythematosus discoides, external irritation as factor in causations of.....*547
erythematosus disseminatus.....71, 82, 332
erythematosus erythema multiforme..... 464
erythematosus, in negress..... 484
erythematosus, of unusual appearance... 84
erythematosus, pathogenesis of.....450, 587
erythematosus, treated with Schamberg's gold solution..... 490
erythematosus, treatment with intravenous injections of gold salts..... 585
follicularis disseminatus fascei..... 88
miliary, of face and scalp..... 78
vulgaris 210, 337, 338
vulgaris exulcerans 351
vulgaris; lupus erythematosus (?) nodular type 87
vulgaris, treated successfully with radium 99
vulgaris, treatment 453
vulgaris, treatment by curettage followed by application of powdered potassium permanganate (method of Auregan).. 324
vulgaris, treatment with tuberculin..... 325
vulgaris, with extensive exacerbation following erysipelas 462

Lymph Glands, syphilitic adenitis..... 337

Lymphangioma? case for diagnosis..... 216
hemangioma and 85

Lymphoblastoma (malignant lymphoma); age and sex incidence, duration and effect of roentgen-ray, radium and surgery 63

Lymphogranulomatosis, Hodgkin's syndrome with meningeal reaction showing lymphocytosis, positive colloidal benzoin reaction and partially positive Wassermann reaction of spinal fluid.. 457
inguinalis *35
lymphoblastoma (malignant lymphoma); age and sex incidence, duration and effect of roentgen-ray, radium and surgery 63
of Schaumann; results after eleven months of intensive treatment with sodium cacodylate 454

Lymphosarcoma *28

McCafferty, L. K.: Hair dyes and their toxic effects; classification and description *136

Schamberg's "peculiar progressive pigmentary disease of skin"..... *53

McGlasson, I. L.: Recurrent erysipelas of legs with dermatitis of feet.....*679

Malaria, therapeutic: See also Neurosyphilis; Paralysis, General
therapeutic, brain after malaria treatment 590
therapeutic, studies of therapeutic effect of fever in experimental rabbit syphilis; thermal death point of spirochaeta pallida *243

PAGE

Markley, A. J.: Point-gap sparkover variations and their effect on dosage in superficial roentgen-ray therapy.....*704
Melanocarcinoma, facial nevocarcinoma, recurring after excision..... 582
Melanoderma, formation of pigment in, of workers in tar, soot, etc..... 450
Melanoma 217, 345
melanotic moles; prognosis..... 728
of arm 742
of eye, in syphilitic rabbit..... 735
pigmentation of type of melanosis of Riehl in locomotive engineer..... 325
primary benign and malignant, of skin. 321
Memmesheimer, A. M.: Significance of cutaneous stimuli for protection of body against infection *4
Meningitis, edematous anthrax of face resulting in 446
Mental deficiency, degree of, resulting from congenital syphilis 727
Mercurials, absorption of, from ointments applied to skin..... 322
proposed method of laboratory evaluation and classification..... 448
Mercury, excretion of, after intramuscular injection of mercuric bromide, inunction and rectal suppositories.....*683
fatal mercurial stomatitis..... 583
Michael, J. C.: Behavior of injected uric acid in patients with eczema.....*308
Critical investigation of relation of end-products of protein metabolism to eczema and kindred disorders.....*294
Michelson, H. E.: Granuloma pyogenicum (correction) 565
Microsporum niveum, tinea microsporica in Sicily 65
Milk, anaphylaxis to..... 329
Miller, H.: Cutaneous manifestations of protein allergy *145
Mineral content of human, dog and rabbit skin 735
metabolism, of skin..... 462
Monilia, black hairy tongue with..... 449
unusual eruption due to organism of Monilia group; preliminary report....*280
Morphea: See Scleroderma
Mount, L. B.: Urticaria pigmentosa.....*715
Mucous membrane, unusual lesions of, apparently due to yeast..... 592
Mycosis: See also Blastomycosis; Ringworm; Trichophytosis, etc.
fungoides.....81, 89, 457, 487, 496, 616
fungoides (tumor stage)..... 90
fungoides, beginning with tumors..... 453
fungoides, granuloma fungoides (tumeurs d'emblée type) 321
fungoides, verrucous and hyperkeratotic type of 449
of nails (?)..... 101
unusual eruption due to organism of Monilia group; preliminary report....*280
unusual skin and mucous membrane lesions apparently due to yeast..... 592
Myelitis, study of 34 cases of rapidly developing syphilitic paraplegia.....*111
Myiasis in Italian Somal 326
Myocarditis, syphilitic, in rabbit..... 198

Nails and endocrines..... 329
family with congenital dystrophy of.... 625
pigmented stripe in.....*533
ringworm of 96, 482
Neoarsphenamine, severe anemia following injections of 455
Nervous System, autonomic, alleged stimulation of, in arsphenamine reactions...*382

PAGE

Neuritis, acrodermatitis chronica atrophicans accompanied by..... 332
Neurodermatitis from oxyuriasis; pathogenesis of prurigo..... 730
Neurofibroma resembling sarcoid of Boeck. 477
Neurofibromatosis.....458, 626, 743
congenital syphilis and..... 323
early and late..... 593
hemorrhage in case of..... 448
incomplete 481
multiple telangiectatic lipomas simulating 452
Recklinghausen's neurofibromatosis and skeleton; plea for complete study of disease *178
with pituitary dysfunction..... 228
Neurosyphilis, conjugal 328
Hodgkin's syndrome with meningeal reaction showing lymphocytosis, positive colloidal benzoin reaction and partially positive Wassermann reaction of cerebrospinal fluid 457
isolated bilateral paralysis of glossopharyngeal nerve due to cerebral syphilis 576
study of 34 cases of rapidly developing syphilitic paraplegia *111
tertiary skin and bone changes in..... 327
treatment by malaria.....*550, 735
treatment, some dogmatic statements concerning 577
treatment, tryparsamide 328
types of, in relation to treatment..... 193
Nevus: See also Angioma
angioma and verrucous nevus..... 230
black mole metastasizing after surgical intervention 324
case for diagnosis..... 94
flammeus, of exensive distribution..... 234
hypertrophic lichen planus or verrucous nevus 354
leukopigmentary, and perinevic vitiligo.. 66
linearis 355
lymphangiomatosus 495
melanotic moles; prognosis..... 728
of sweat glands..... 463
pigmented hairy nevus, with growth on eyeball 346
pigmentosus? Case for diagnosis..... 493
pigmentosus et pilosus treated with roentgen rays and radium..... 353
pruritic nevus unius lateris..... 625
removed by fulguration..... 610
Newspaper ink dermatitis..... 472
Nicholas, H. O.: Behavior of injected uric acid in patients with eczema.....*308
Nickel, experimental idiosyncrasy to..... 739
Nodules, juxta-articular, in a syphilitic... 588

OBITUARIES:
Alembert Winthrop Brayton, 1848-1926. 568
Edward Bennett Bronson, 1843-1925.... 188
James Moore King, 1867-1926..... 570
William Burwell Trimble, 1870-1925..... 566
Ochronosis; varicose ulcers..... 335
Oil acne in lathe workers..... 459
Ointments applied to skin; absorption of mercurials from 322
O'Leary, P. A.: Treatment of neurosyphilis by malaria *550
Oliver, E. L.: Treatment of pruritus ani by electric ionization.....*560
Oriental Sore: see Leishmaniasis
Oroya Fever, etiology of..... 586
Oxyuriasis, neurodermatitis from; pathogenesis of prurigo..... 730

Paget's disease of breast..... 459
Papilloma treated by electrocoagulation... 342

PAGE

Paralysis, isolated bilateral, of glossopharyngeal nerve, due to cerebral syphilis... 576
Paralysis, General, and congenital syphilis in children..... 456
incipient; study of earliest symptoms presented by 74 cases..... 319
malaria in treatment of.....200, 202, 456, 586, 589, 732
malaria treatment, brain after..... 590
postmalaria condition of cerebrospinal fluid in 587
Paraplegia, syphilitic, rapidly developing; study of 34 cases.....*111
Parapsoriasis.....208, 330, 360, 361, 744
guttate 330
xanthoma with erythema telangiectodes and 622
Pardo-Castello, V.: Congenital partial albinism *173
Lymphogranulomatosis inguinalis..... *35
Parker, S. T.: Treatment of neurosyphilis by malaria *550
Peas, primary allergy and shock from.... 329
Peck, S. M.: Granuloma inguinale; clinical observations, vaccine treatment and allergic reactions *14
Pellagra 622
case for diagnosis..... 492
Pels, I. R.: Unusual eruption due to organism of Monilia group; preliminary report *280
Pemphigus 82, 217, 601
neonatorum, possible cause for..... 327
postvaccinial 358
Penis, angioneurotic edema of scrotum and Fordyce's condition of..... *50
Phenobarbital, atrophy of tongue from.... 223
Phenolphthalien eruption.....100, 235, 477
Phlebitis, subacute syphilitic, in acute gonorrhea 452
Pigmentation and keratoderma of arsenical origin 451
of lips and buccal mucosa; argyria?.... 493
pigmented stripe in nail.....*533
Piness, S. G.: Cutaneous manifestations of protein allergy.....*145
Pityriasis capitis, liquor of Alibour (solution of copper sulphate in water) in treatment of..... 581
rosea, etiology of..... 455
rubra pilaris....205, 219, 231, 232, 491, 585
rubra pilaris? Linear lichen planus? Case for diagnosis..... 476
steatoides, treatment by solution containing sulphur and oil of cade..... 449
Pityrosporon of Malassez, study of dandruff and *270
Poikiloderma 595
atrophicans vasculare 594
reticulated pigmentary, of face and neck 580
Polyarthritis in syphilitic child..... 202
Pregnancy, treatment of syphilis during.. 582
Prizes for research on venereal disease 443, 573
Procaine; absorption of ultraviolet rays in human skin..... 730
inhibition by procaine of light dermatitis and pigmentation..... 730
Protein allergy, cutaneous manifestations..*145
metabolism, relation of end-products of, to eczema and kindred disorders.....*294
Prurigo lymphadenique..... 456
and scratching..... 450
nodularis 624
pathogenesis of..... 730
summer, of Hutchinson, in twins..... 625

PAGE

Pruritis ani and pruritis vulvae of mycotic origin..... 580
ani, treatment by electric ionization....*560
in diabetic, intramuscular injections of insulin in 490
pruritic nevus unius lateris..... 625
treatment of circumscribed pruritus (neurodermite) by ionization with aconitine 453
vagotonia; pruritus 607
Psoriasis and diabetic heredity..... 454
case for diagnosis..... 96
follicular 83
insulin and 454, 454, 456
of palms 490
treated with Alpine lamp..... 338
treatment with potassium and sodium tartrobismuthate 325
treatment with quartz lamp..... 576
vulgaris; results of cytologic investigations 326
Purpura annularis telangiectoids, etiology of 461
hemorrhagica, idiopathic treatment with mercury vapor quartz lamp..... 447
pretuberculous rheumatoid 329
Pyoderma and dermatitis vegetans..... 212

Radium applicator for treatment of lesions about head, face and neck..... 193
emanation, effect on epithelioma of floor of mouth 223
Rat-Bite Fever, organism causing, experimental studies with spiral organism found in wild rat and identical with.. 320
Rauschkolb, J. E.: Excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683
Rose bengal test for liver function; its use in therapy of syphilis.....*122
Raynaud's Disease.....95, 360, 362
chronic acro-asphyxia, scleroderma and.. 68
Recklinghausen's Disease: See Neurofibromatosis
Rectum, hyperplastic infiltration of, with fibrous stricture 449
Rhinophyma, histopathology and treatment of 586
in woman 86
Righter, L. L.: Distinctive and severe form of erysipeloid among fish handlers; clinical and laboratory studies; demonstration of bacillus of swine erysipelas *662
Ringworm: See also Favus
allergic reactions in cases of cutaneous mycosis 585
demonstration of cultures and photographs of some dermophytes..... 214
of nails 96, 482
thallium in treatment of..... 584
tinea favosa of scalp..... 233
tinea microsporica in Sicily..... 65
tricophytine in diagnosis and treatment of 584
Roentgen Rays, action of rays on cells.... 201
alopecia following treatment with..... 339
burn, with sun lamp therapy..... 346
burns, deep, treatment by excision and tissue shifting 192
cancer, vitamin imbalance, and roentgen-ray activity 447
dermatitis, chronic radiodermatitis..206, 474
effect of, on division rate of paramecium 320
favus after treatment with..... 338
hard and soft, biologic action of, in relation to skin cancer..... 730
nails, treatment of..... 448
skin reactions to alternate heat and x-ray exposures 320
therapy, in skin diseases..... 202
therapy, superficial, point-gap sparkover variations and their effect on dosage in *704
ulcer, treatment of..... 590

Rose Bengal test: See Liver PAGE

Rosen, I.: Skin manifestations seen in a general hospital....*693

Rule, A. M.: Studies of therapeutic effect of fever in experimental rabbit syphilis, thermal death point of Spirochaeta pallida *243

Sachs-Georgi and Wassermann reactions in leprosy 67

Salinger, R.: Unusual eruption due to organism of Monilia group; preliminary report *280

Sanocrysin exanthem 737

Sarcoid 85, 491, 494
cutaneous 66
Darier-Roussy type....333, 361, 461
massive fibrous or fibroconnective hypodermic sarcoid 324
of Boeck 100, 491
of Boeck, neurofibroma resembling.... 477
Spiegler-Fendt 232
tuberculous lupoid 461
type of, approaching erythema induratum of Bazin 461

Sarcoma: See also Lymphosarcoma
Kaposi's 90
multiple hemorrhagic (Kaposi).... 614
of caruncle 446
sarcoma 349

Scarlatinal infection due to streptococcus viridans 448

Schamberg, J. F.: Studies of therapeutic effect of fever in experimental rabbit syphilis; thermal death point of Spirochaeta pallida *243

Schamberg's Disease: See Dermatoses, progressive pigmentary

Schreiber, N.: Excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683

Sclerema neonatorum *635
neonatorum; chemistry of subcutaneous tissues 732

Scleroderma 208, 343, 467, 481, 743
and sclerodactylia 204
chronic acro-asphyxia, Raynaud's disease and 68
en bande; tertiary syphilis.... 233
guttate morphea (?).... 71
localized 335
lupus erythematosus diffuse scleroderma. 465
syphilis and 450
with diabetes mellitus, controlled by insulin 613

Sclero-edema in new-born.... 737

Scrofuloderma 87

Shaffer, L. W.: Calcification of skin, including epiderm, in connection with extensive bone resorption....*503

Skin, action of intradermal irritation on threshold for stimuli.... 729
elastic degeneration of.... 91
grafts, difference in evolution of pediculated and transported, in facial lupus vulgaris 462
in children 738
lesions, and cutaneous gland secretions.. 739
manifestations, seen in general hospital..*693
mineral content of human, dog and rabbit skin 735
mineral metabolism of.... 462
reactions of, to alternate heat and x-ray exposures 320
significance of cutaneous stimuli for protection of body against infection.... *4

PAGE

Smallpox and unvaccinated friends.... 571
blood of patients with, during recent epidemic 576
embargo removed 444
epidemic in Kentucky.... 724
in Texas increasing.... 724
record in Syracuse.... 724
vaccination, erythema multiforme complicating 64

Smith, D. K.: External irritation as factor in causation of lupus erythematosus discoides *547

Societies, foreign dermatologic....
....62, 191, 318, 445, 575, 726
local dermatologic (officers)....
....61, 190, 317, 444, 574, 725
national dermatologic (officers)....
....190, 316, 444, 574, 725
national, fiftieth anniversary meeting of American Dermatological Association.. 316
national, founding of American Dermatological Association *371

Society Transactions:
American Dermatological Association.... 597
Brooklyn Dermatological Society....204, 349
Chicago Dermatological Society..69, 209, 464
Detroit Dermatological Society....491, 624
Manhattan Dermatological Society.... 474
Minnesota Dermatological Society.... 740
New England Dermatological Society.... 340
New York Academy of Medicine, Section on Dermatology and Syphilis..76, 214, 335
New York Dermatological Society....
....97, 229, 354, 482
Philadelphia Dermatological Society....
....90, 224, 330, 479
Pittsburgh Dermatological Society....
....94, 359, 591

Sodium, thiosulphate, in arsphenamine dermatitis 65

Sollmann, T.: Excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683

Specialties, hospitals providing approved residencies in 572

Spider poisoning; arachnidism.... 193

Spirochaeta, pallida, thermal death point of, studies of therapeutic effect of fever in experimental rabbit syphilis..*243

Sporotrichosis 192

Staphylococcic pustule, ikonographic study of, and lesions derived from it (sycoses) 451
reducing power of, as element of prognosis of pyodermas and in selection of stock vaccines 458

Stomatitis, fatal mercurial.... 583
is aphthous stomatitis due to virus of herpes simplex? *439
preepitheliomatous vegetating 323

Streptococcus, occurrence of, on healthy and diseased skin.... 731
viridans, scarlatinal infection due to.... 448

Sulphur, solution containing oil of cade and, in pityriasis steatoides.... 449

Suppositories, excretion of mercury after intramuscular injection of mercuric bromide, inunction and rectal suppositories *683

Suprarenals, tumors, cortical, cutaneous striae, purpura, high blood pressure, amenorrhea, and obesity connected with hypo-adrenalism and dysinsulinism of acquired or congenitally syphilitic origin; new pluriglandular syndrome.. 65 324

Sweat Glands, nevus of.... 463

Sycosis vulgaris cured with intradermal vaccine injections 207

Symbiosis, importance of.... 446

PAGE

Sympatheticotonia, eczema 610
Syphilids, ecthyma; syphilitic ulcers..... 88
frambesiform; primary and secondary syphilis 602
maculopapular, arsphenamine hepatitis... 474
miliary papular syphiloderm..... 221
nodular, arranged in large circles..... 454
palmar and plantar, in teritiary syphilitic 87
scaling serpiginous, of palms and soles.. 741
traumatic ulcerative, of foot..... 582
vitiligoid 88
Syphilis 739
acquired, in congenital syphilitic?..... 221
adenitis in 337
angioneurotrophic endocrines of, especially erythromelie (Pick)..... 451
arsenical reactivation of..... 462
campaign against, in Belgium.....199, 442
cardiovascular 573
chancre of eyebrow (right side), with secondary lesions 89
congenital 577
congenital, and general paralysis in children 456
congenital, hypo-adrenalism and dysinsulinism of acquired or congenitally syphilitic origin; new pluriglandular syndrome 324
congenital, kidney tumor with..... 738
congenital, late, of second generation affecting several members of same family 458
congenital, localized dystrophies of..... 452
congenital, mental deficiency resulting from 577, 727
congenital, pathogenesis of..... 583
congenital, polyarthritis in syphilitic child 202
congenital, Recklinghausen's disease and. 323
congenital, sabre tibias in very late case of 582
congenital, 25 years of, in Boston..... 577
conjugal, of nervous system..... 328
contagion before appearance of chancre; effect of preventive treatment..... 454
control of venereal diseases..... 576
control, rural organization for, in region of lower Seine..... 456
diabetes in 66
diagnosis, case for; erythema multiforme or syphilis? 340
diagnosis, error of basing on Kahn reaction alone 64
diagnosis, microscopic slide precipitation test for syphilis; preliminary report... 63
diagnosis, new serum diagnosis of syphilis 586
diagnosis, serodiagnosis of frambesia and syphilis 732
early history of..... 203
ecthyma; syphilitic ulcers..... 88
exopthalmic goiter and..... 323
familial localizations of..... 200
hyperplastic infiltration of rectum with fibrous stricture in..... 449
immunologic relations between frambesia and 732
in gynecologic clinic at Rio de Janeiro... 590
in maternity clinic of Toulouse, serologic statistics 455
in third generation from ophthalmologic standpoint 458
increase of..... 734
juxtra-articular nodules in..... 588
latent 201
lichen scrofulosorum (?) or secondary follicular syphilis or latent syphilis.. 601
melanoma of eye in syphilitic rabbit.... 735
multiple visceral, double syphilitic mastitis in course of..... 323
of aorta and heart.....202, 577
pernicious anemia; glossitis..... 222
reinfection 323, 455
resistant ulcerating 69
rose bengal test for liver function; its use in therapy of syphilis.....*122

Syphilis—Continued PAGE

scleroderma and 450
superinfection 88, 339
superinfection of husband and wife, probable 81
tertiary, of face with firm, noninflammatory form of infiltration of lymphodermic type 451
tertiary; scleroderma en bande..... 233
tertiary, tubercular, of nose, with congenital syphilis; massive hyperostosis of both tibias..... 459
testes from the syphilitic, study of..... 328
therapeutic effect of fever in experimental rabbit syphilis; thermal death point of Spirochaeta pallida.....*243
traumatic 461
treatment 576
treatment, according to Jacques de Bethencourt, 1527; extracts from French translation by Alfred Fournier, 1871 .....*427
treatment, during pregnancy..... 582
treatment, llama serum in..... 590
treatment of early syphilis.....733, 733
treatment, protein 200
tuberculosis in 588
urobilinogen determination in treated syphilitic patients *434
Syphilology, graduate courses in medical subjects and residencies in specialties. 572
index to current literature.....108, 241, 367, 500, 632, 752
Syringomyelia, unilateral 608

Tabes appearing 5 years after chancre in patient aged 27..... 583
combined, in woman of 24 years..... 452
Tar, formation of pigment in the melanoderma of workers in..... 450
Telangiectasis, generalized; etiology and pathology *387
hereditary hemorrhagic *1
superficial, of lower part of legs..... 321
Templeton, H. J.: Dandruff and pityrosporon of Malassez.....*270
Is aphthous stomatitis due to virus of herpes simplex? *439
Pigmented stripe in nail.....*533
Testes, study of, from syphilitic..... 328
Thallium acetate, epilation by, in fungous diseases of hair..... 463
epilating properties of..... 584
in treatment of tinea..... 584
Thermalgia 612
Thienes, C. H.: Alleged stimulation of autonomic nervous system in arsphenamine reactions *382
Thrombo-angiitis obliterans 470
Tinea: See Ringworm
Tongue, atrophy of, from phenobarbital.... 223
black hairy tongue with monilia..... 449
case for diagnosis..... 94
Trichophytine in diagnosis and treatment of tinea 584
value of cutaneous reactions to..... 325
Trichophytosis; case for diagnosis..... 608
due to faviform trichophyton..... 196
erythematosquamous, of bearded region.. 457
Trichotillomania *59
Tuberculids, atrophic papulosquamous..... 460
papulonecrotic 496
papulonecrotic; case for diagnosis..... 229
papulonecrotic; folliclis type..... 362
recurrent papular eruption; case for diagnosis 229
tuberculosis and 211
Tuberculin therapy, failure of, in 4 cases of cyanotic puffiness of extremities.... 460
Tuberculoma, massive and fistulous fibrous, of hypoderm 324

PAGE

Tuberculosis and tuberculids..... 211
case for diagnosis.....342, 597
cutis 496, 496, 615
cutis, unusual form of..... 491
erythema nodosum and..... 200
hypertrophic, papillomatous, nonulcerative, of tongue..... 458
in syphilis 588
of eye 446
pathogenesis of angiolupoid..... 327
verrucosa cutis.....207, 356, 473, 496
Turpentine, occupational dermatitis from.. 319
Twins, summer prurigo of Hutchinson in.. 625

Ulcers, causes of chronicity of, of leg and general method for their treatment.... 459
chronic, of penis; case for diagnosis..... 486
of leg 94
ulcus vulvae acutum and herpes vulvae.. 722
varicose 83
varicose; ochronosis 335
Ultraviolet Rays, absorption of, in human skin 730
inhibition by procaine of light dermatitis and pigmentation 730
standardization of; ultraviolet unit..... 63
Uric Acid, injected, behavior of, in patients with eczema *308
Urobilinogen determination in treated syphilitic patients *434
Urticaria 74
alkali reserve and..... 328
pigmentosa.....97, 97, 97, 99, 195, 476, 476, 482, 494, 593, 614, 614, *715
psychic 67

Vaccine Therapy of acrodynia..... 736
Vaccines, reducing power of staphylococci as element of prognosis of pyodermas and in selection of stock vaccines.... 458
Vagotonia; pruritus 607

PAGE

Venereal Disease clinic in Mobile..... 571
postgraduate course on..... 571
prizes for research on.....443, 573
Verruca, common warts of mouth..... 736
plana; case for diagnosis..... 79
planae juveniles 84
sulpharsphenamine in treatment of..... 727
Vitamin imbalance, cancer, and roentgen-ray activity 447
Vitiligo 497
leukoderma syphiliticum 192
of symmetrical distribution, with extensive alterations of sensitivity of bone to vibrations 582
perinevic and leukopigmentary nevus.... 66

Wassermann Reactions, daily variations in complement fixation (Kolmer) and precipitation (Kahn) reactions in untreated syphilitic patients..... 578
local, made with serum from the lesion; value in diagnosis..... 451
reactivation of, under infection..... 588
Sachs-Georgi and, in leprosy..... 67
Wax, epilating, recipe for..... 579
Weidman, F. D.: Calcification of skin, including epiderm, in connection with extensive bone resorption.....*503
Williams, C. M.: Hereditary hemorrhagic telangiectasia *1

Xanthoma, cholesterinemia and glycemia in course of; insulin therapy..... 458
tuberosum 90
tuberosum multiplex.....322, 496, 592
versus gout 737
with erythema telangiectodes and parapsoriasis 622
Xeroderma pigmentosum.....226, 604

Yeast, unusual skin and mucous membrane lesions apparently due to..... 592

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CONTENTS

PAGE

Sclerema Neonatorum. A. M. H. Gray, M.D., F.R.C.P., London 635

Disseminated Palmar and Plantar Keratoderma: Report of a Case. David Lieberthal, M.D., Chicago 655

A Distinctive and Severe Form of Erysipeloid Among Fish Handlers: Report of Clinical and Laboratory Studies; Demonstration of the Bacillus of Swine Erysipelas. Joseph V. Klauder, M.D.; Linwood L. Richter, M.D., and Malcolm J. Harkins, V.M.D., Philadelphia 662

Recurrent Erysipelas of the Legs with Dermatitis of the Feet. J. Le McGlasson, M.D., San Antonio, Texas 679

Excretion of Mercury After Intramuscular Injection of Mercuric Bromide, Inunction and Rectal Suppositories. H. N. Cole, M.D.; John A. Gammel, M.D.; J. E. Rauschkolb, M.D.; Nora Schreiber, B.S., and Donald Seelmann, M.D., Cleveland 683

Skin Manifestations Seen in a General Hospital. Hermann Goldenberg, M.D., and Isadore Rosen, M.D., New York 693

Point-Gap Sparkover Variations and Their Effect on Dosage in Superficial Roentgen-Ray Therapy. A. J. Markley, M.D., Denver 704

Fixed Arsphenamine Dermatitis. Henry H. Hazen, M.D., Washington, D.C. 713

Urticaria Pigmentosa. Louis B. Mount, M.D., Albany, N.Y. 715

Editorials:

Ulcus Vulvae Acutum and Herpes Vulvae 722

News and Comment 723

Abstracts from Current Literature 727

Society Transactions:

Minnesota Dermatological Society 740

Book Reviews 747

Index to Current Literature 749

General Index 755